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Vol. VIII, No. 1 February, 1939

DIAPHRAGMATIC HERNIA

Report of Cases Illustrating Its Varieties

PHILEMON E. TRUESDALE, M. D.
Fall River, Massachusetts

FEW developmental defects are as perplexing as diaphragmatic hernia. In fact, this anomaly appears in so many forms that it defies simple classification. The variety found in children, for example, differs from that affecting adults in almost every important aspect. The congenital type of hernia observed in early life is usually bizarre and relatively rare. On the other hand, esophageal hiatus hernia, which develops at the meridian of life or later, is by far the most common type of diaphragmatic hernia. Its cause is a relaxation of the muscle structure of the diaphragm superimposed upon a congenital deficiency of tissue surrounding the lower end of the esophagus. As a result of the atonic muscle support at this point, any increase of intra-abdominal pressure may force the cardiac end of the stomach into the mediastinum. Some patients with hiatus hernia go through life without symptoms. In others, however, the condition may be so embarrassing and complaints so persistent that surgical intervention is warranted. In many cases symptoms are mistaken for those of peptic ulcer, cholecystitis, and angina pectoris. This form of hernia and the congenital form found in children comprise two great divisions of diaphragmatic hernia.

ANATOMIC CONSIDERATIONS

Any study of the diaphragm must be based on a knowledge of its anatomic structure. Figure 1 shows the thoracic surface of the diaphragm with its muscle structures, its attachments to the ribs, and the openings through which pass the aorta, the vena cava, and the esophagus.

Read before the Postgraduate Surgical Assembly, the ninth annual meeting, of The Southeastern Surgical Congress, Louisville, March 7, 8 and 9, 1938.

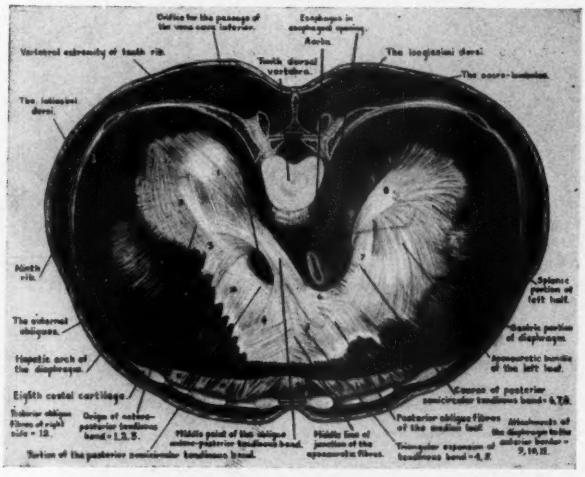

Fig. 1. Thoracic surface of diaphragm.

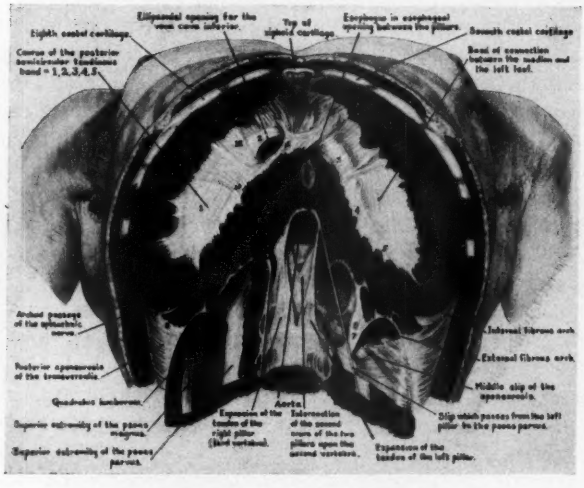

Fig. 2. Abdominal aspect of diaphragm.

Figure 2 shows the abdominal aspect of the diaphragm. The powerful attachments of the tendinous portions of the diaphragm to the first four lumbar vertebrae are seen from below. The esophageal opening appears between the pillars. The openings for the aorta and vena cava are also seen.

Blood Supply

The blood supply of the diaphragm is derived from branches of the internal mammary blood vessels, the superior and inferior phrenic arteries which spring from the abdominal aorta. Figure 3 shows the arteries which supply the superior surface. The internal mammary blood vessels descend to the diaphragm, spreading out over its central and anterior surface and anastomosing with the intercostal vessels. Some vessels are seen on the upper surface of the diaphragm; others become embedded in the muscle.

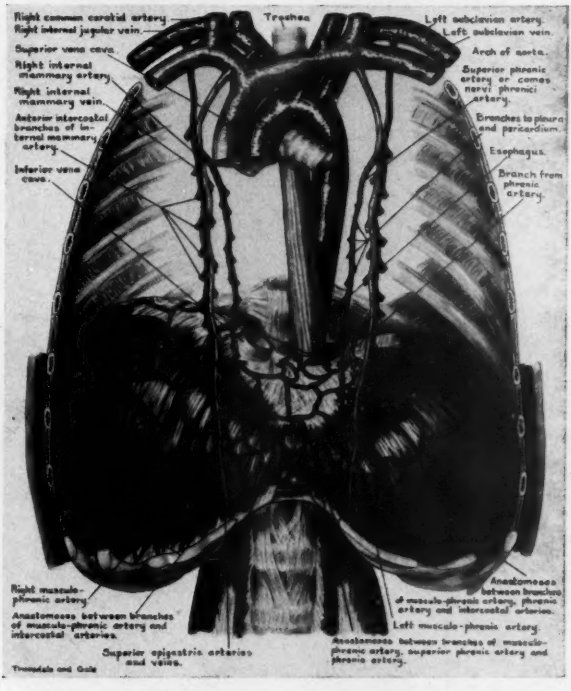

Fig. 3. Blood supply of diaphragm, superior surface.

Figure 4 shows the blood supply of the inferior surface of the diaphragm. Arising from the abdominal aorta, the inferior phrenic arteries are the principal source of blood supply to the muscular portions of the diaphragm. They ascend along the crura and reach the central posterior edge where they subdivide, and form an intricate network. Branches of these arteries and veins penetrate to the suprarenal glands, the liver, stomach and esophagus.

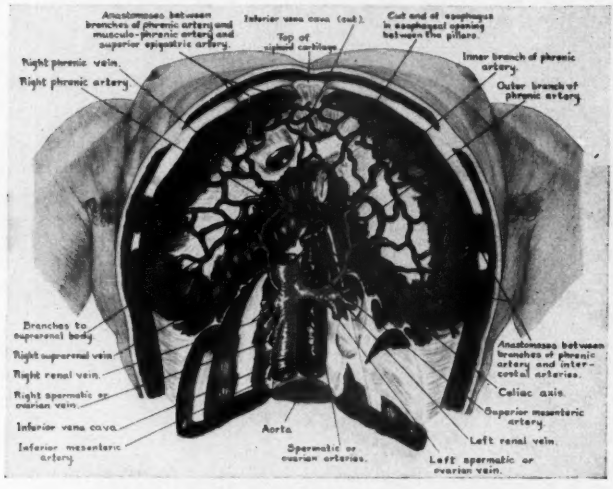

Fig. 4. Blood supply of diaphragm, inferior surface.

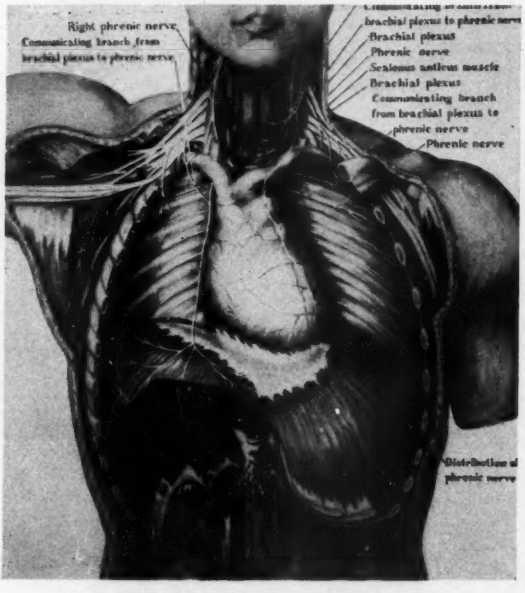

Fig. 5. Innervation of the diaphragm.

Innervation

Figure 5 shows the innervation of the diaphragm. The chief nerve is the phrenic which has its origin from the fourth cervical nerve. It pursues an anterolateral course and pierces the diaphragm

at the junction of the musculature with the central tendon. The left phrenic nerve is longer than the right owing to the greater inclination of the heart toward the left and to the fact that the left leaf of the diaphragm is somewhat lower than the right. The right phrenic nerve extends inward along the innominate vein, the superior vena cava, and the pericardium; the left phrenic passes over the arch of the aorta, the left ventricle and the cardiac branch of the left vagus nerve. Extensive experimentation has shown that the *motor* supply of the diaphragm is from the phrenic nerve alone. The intercostal

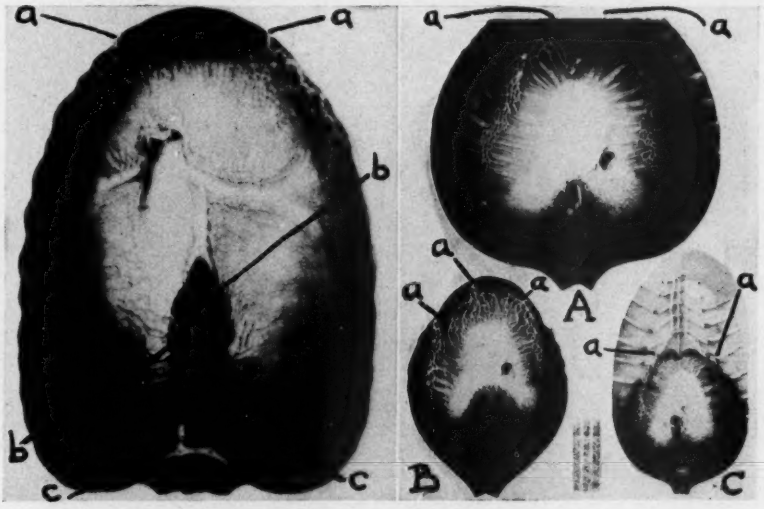

Fig. 6. Lymphatics of the diaphragm of the horse.
Fig. 7. Lymphatics of the diaphragm in man, A, a dog, B, and a rabbit, C.

nerves pass through the diaphragm without giving off branches to the diaphragmatic muscle. Troitzky[1] after dissecting the nerves supplying the diaphragm in 130 cadavers and performing experimental neurotomy on 65 dogs was convinced that the phrenic nerve was the sole motor supply to the diaphragmatic musculature. The lower six thoracic intercostal nerves supply afferent sensory fibers.

Lymphatics

The diaphragm of the horse, as injected by Sappey,[2] owing to its enormous development, provides a good illustration of the origin of the lymphatic network covering the muscle fibers and accompanying them to the lymphatic trunks into which they drain (fig. 6). They comprise two anterior groups *a, a,* five trunks which lie centrally and give off branches that communicate with glands located around the vena cava, *b, b,* and five or six aortic trunks which lie posteriorly on either side, *c, c.*

Figure 7 illustrates the lymphatic system in the diaphragm of a man A, a dog B, and a rabbit C. In the rabbit the lymphatic system is as highly developed as in the dog and the arrangement is similar, even in the anterior portions. In man, however, the two anterior groups, *a, a,* continue in closer relation with the mammary veins. In addition, one or two independent vessels are found between the two groups of main trunks which empty into the same vessels. In observing the myriads of small vessels contiguous and superimposed, one is surprised to find so many vessels in so thin a membrane.

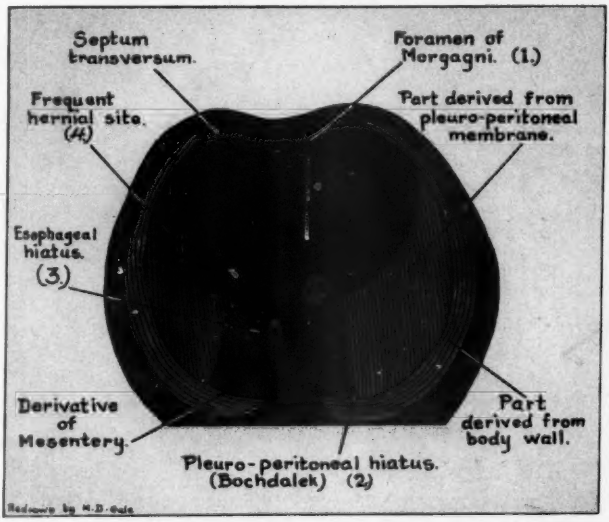

Fig. 8. Elements composing the diaphragm. From Prentiss, *op cit.* p. 196, fig. 191.

The vessels of the inferior surface of the diaphragm are especially well developed. They traverse both muscular and tendinous portions of the diaphragm, uniting the network above with that below. Both systems are exceedingly full and closemeshed in the region of the central tendon. There is almost a complete break at the attachment of the suspensory ligament, thus creating a kind of separate lymphatic system for each leaf of the diaphragm. The muscular portions not covered with peritoneum have no lymphatic vessels.

After the lymph is carried to the lymph nodes of the diaphragm, it passes on to the anterior and posterior mediastinal nodes and to the lower sternal glands. Some collecting stems pass backward medially toward the aortic opening, beyond which they pass and finally terminate in the upper celiac nodes. From this point the course of the lymph is variable. It passes through the superior and

inferior network to join other lymphatics emptying into the thoracic duct, and finally empties into the subclavian or innominate veins.

The lower network is in close relation with the lymphatic system of the abdomen, especially the liver; the upper plexus is intimately connected with the thoracic lymphatics. Since both these networks are closely intertwined, an infection on one surface of the diaphragm can easily reach the other by way of the lymphatics. This intercommunication of the lymphatic system assumes greater importance in dealing with infections in this region.

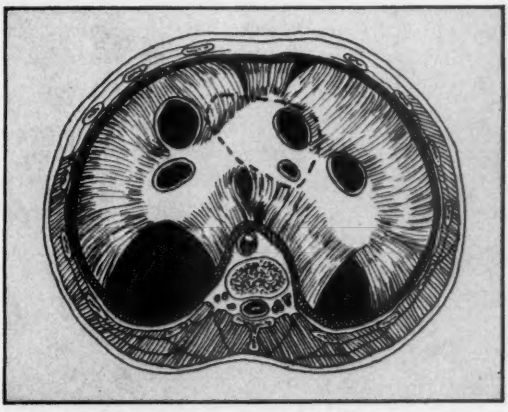

Fig. 9. Composite drawing showing various sites of herniation.

Thus in anomalies and infections of the diaphragm, we must expect to find some degree of embarrassment of the three great vital functions of circulation, respiration, and digestion.

Sites of Herniation

Herniation through the diaphragm usually occurs in weak areas in its structure, resulting from lack of fusion before birth. The diaphragm is made up of four sources: (fig. 8) the rudimentary septum transversum; the pleuroperitoneal membranes; the posterior central portions derived from the dorsal primitive mesentery; and the mesentery of the body wall. Failure of fusion of any of these parts furnishes a point of weakness through which herniation may occur.

The most common site of herniation in the congenital cases is the foramen of Bochdalek (fig. 2) situated posteriorly. Other sites are the foramen of Morgagni, the esophageal hiatus, and the central tendon. Figure 9 is a composite drawing showing the various sites of herniation in our series of both congenital and traumatic cases of diaphragmatic hernia.

General Considerations

The criteria upon which to judge the application of surgery in cases of hiatus hernia have not been clarified, nor can they be laid down upon the basis of our present knowledge. Dr. Bremer, professor of embryology at the Harvard Medical School, has said that there is as yet no precise knowledge as to just what course the various structures pursue during the embryonic vagrancies associated with the descent of the diaphragm. Prentiss,[3] Toldt,[4] and Piersol[5] have shown that the position of the diaphragm changes at different months of fetal growth and the septum and adjacent structures are moving unceasingly for many weeks to their permanent location. Therefore, it follows that knowledge concerning the development of the diaphragm, of which we are so much in need, is, as yet, only fragmentary.

To illustrate some of the complexities of diaphragmatic hernia, I have selected several cases, each of which manifests a form of hernia differing from the others. The first case is one of congenital diaphragmatic hernia in a girl 4 years old. She was my fourteenth case in children, and to my surprise the conditions found at operation differed entirely from those in any of the others. First, it was a right-sided diaphragmatic hernia. Second, the arrangement and manner of transposition of the herniated viscera together with the defects discovered in the diaphragm were so involved that it was difficult to determine what changes were developmental and what influences produced the extraordinary shifting of structures in prenatal and postnatal life. In such cases the mechanism may be so intricate as to defy accurate interpretation, even at operation or autopsy.

This child was admitted July 15, 1935. Born at term, she was the second child of normal parents. The birth weight was four pounds, twelve ounces; the infant was undernourished and looked premature. Her extremities were limp and she had a dry, irritating cough and refused nourishment. Liquids were given by gavage. Until the age of six weeks she vomited most of the feedings but was able to retain diluted skimmed milk. By the age of four months she was eating cereals, orange juice and viosterol in addition to milk. At this time she weighed eight pounds.

When five months old the right arm and left leg began to increase in size. Two months later the baby was admitted to Johns Hopkins Hospital where Dr. T. C. Goodwin on physical examination made the diagnosis of right congenital diaphragmatic hernia. X-ray examination revealed the transverse colon and loops of small intestine in the right pleural cavity. Operation was not advised at that time because of the child's retarded development. Her weight remained

almost stationary. She appeared undernourished and frail. Edema of the right arm and left leg persisted. She vomited frequently, and suffered from chronic bronchitis. From the age of 2½ years to 4 she gained only 1½ pounds.

Fig. 10. Drawing from x-ray films. Anterior view of herniated viscera—cecum, ascending colon, portion of transverse colon, and a large portion of the small intestine in right thoracic cage. The heart is somewhat displaced to the left. The stomach is in normal position below the diaphragm.

She came to our Clinic at the age of four years, anemic, emaciated, and asthenic. Her weight was 22 pounds. She was pigeon-breasted. Rumblings and gurglings were audible over an asymmetrical, bulging right thorax. Breath sounds on the right were limited to the area above the third rib. The apex of the heart was in the left mid-axillary line. The right arm and left leg were swollen moderately. The intellect was that of a child of two years or less. The diagnosis agreed with that already made, congenital diaphragmatic hernia in the right leaf of the diaphragm.

X-ray examination July 16, 1935, revealed the stomach in normal position, but hypotonic and moderately dilated. Figure 10 is a copy of the x-ray film showing an anterior view of the herniated viscera. The cecum, ascending colon, portion of the transverse colon and a

large portion of the small intestine were observed in the right thoracic cage, compressing the right lung and displacing the heart toward the left.

Further examination showed the intestines entering the thorax through the anterior portion of the right leaf of the diaphragm. Therefore it seemed that laparotomy offered a more ready access to the hernia.

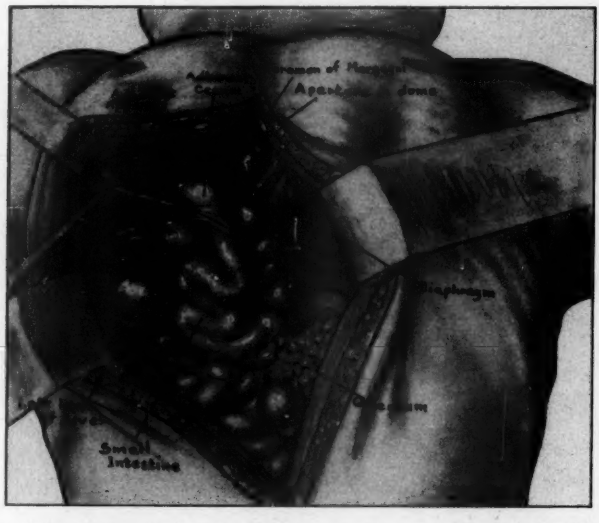

Fig. 11. The apertures in the diaphragm are seen from below. The right lobe of the liver with its attachments and the appendix and cecum are seen occupying the hernia sac in the dome of the right diaphragm.

October 26 the abdomen was opened by a vertical incision through the right rectus muscle. A curious arrangement of the structures in the upper abdomen and right chest was brought into view (fig. 11). The cecum, appendix, small intestines, transverse colon, piece of omentum and right lobe of the liver were seen in a huge sac in the right dome of the diaphragm which had reached the level of the second rib. The cecum and appendix appeared to be adherent to the serosa of the hernial sac and to structures which occupied a higher position in the sac. The appendix was at the extreme right firmly adherent to the thoracic wall.

Adhesions binding cecum and appendix were separated, the diseased appendix removed and the cecum replaced in the abdomen. The small intestines and colon were free and were easily reduced. When these hollow viscera had been brought down into the abdomen, it was noted that there were several openings in the right leaf of the diaphragm, varying considerably in size and each forming a

true hernia. The smaller sacs were nearer the central tendon, whereas the largest, which had contained appendix, cecum, large and small bowel, occupied the major portion of the dome of the diaphragm. In the summit of this sac was found an aberrant hepatic left lobe, normal in color and relatively small. It had migrated to

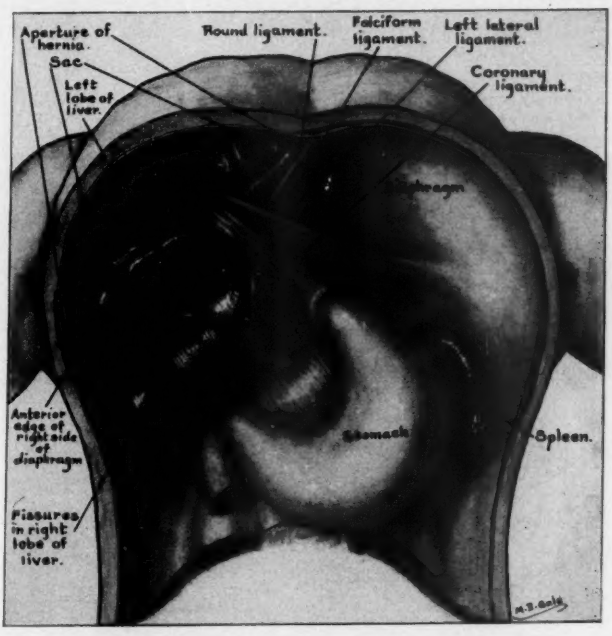

Fig. 12. Aberrant left lobe of liver with abnormally stretched ligaments.

the right side and rotated upward on the falciform and coronary ligaments, drawing them taut. These ligaments, abnormally stretched, permitted the left lobe to become firmly adherent to the serosa of the large sac, the summit of which reached the level of the second rib (fig. 12). A somewhat similar case was reported in 1935 by Suarez of Buenos Aires.[6]

At the first operation an attempt to remove this left lobe of the liver was unsuccessful because of its high position and the assumed density of its attachments. After reduction of other structures, each sac was closed by plication. Then with interrupted silk sutures the muscular margins of the diaphragmatic opening were brought into apposition and sutured with silk. The foramen of Morgagni, which was larger than normal, was closed with silk. This hernia, therefore, was not through the foramen of Morgagni, but involved almost the entire anterior portion of the right diaphragm, as if continual pressure of the viscera had created a huge thin sac and two smaller sacs

out of what was once the right leaf of the diaphragm, the anterior border of which had failed to fuse with the thoracic wall. It was regretted that the main breach in which the liver lobe was attached could not be dealt with safely at this time. Further efforts to dislodge the aberrant structure, no doubt, would have greatly increased the operative risk. The laparotomy wound was closed completing the first stage of what eventually provide to be a more intricate affair.

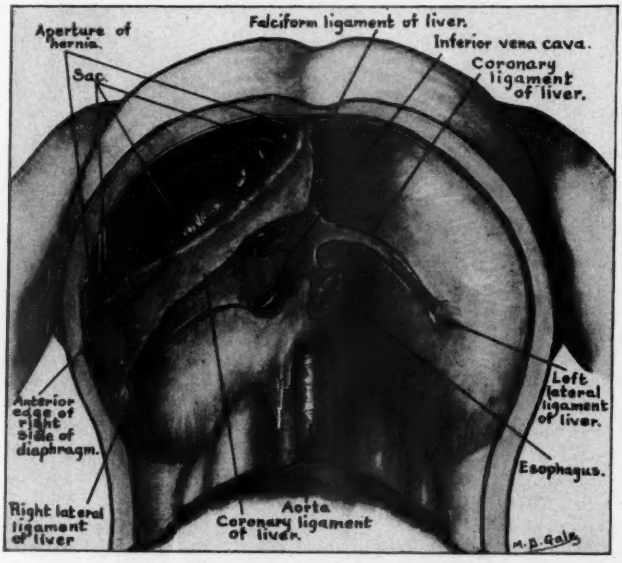

Fig. 13. View of anterior surface of diaphragm seen from below, showing failure of fusion of anterior margin of the diaphragmatic leaf with the thoracic wall over more than a third of its perimeter.

The immediate postoperative recovery was good. On the third day the temperature dropped to normal and the child's condition remained satisfactory for two weeks. Then she began to refuse food and to lose weight. X-ray examination with barium enema on November 13, eighteen days after operation, showed the colon pursuing a fairly normal course in the abdomen; several loops of terminal ileum, however, had passed upward to occupy a position high in the right chest. The heart was slightly displaced to the left. There were no symptoms of intestinal obstruction. She was up and about, walking with help.

Two weeks later her condition showed no improvement. Therefore on November 27 under gas-oxygen ether anesthesia the abdomen was opened by an incision parallel to the right costal border. In the anterior portion of the diaphragm a large thin hernial sac was again exposed containing loops of small intestine which were

adherent to the perimeter of the sac. In the extreme upper portion was the aberrant lobe of liver firmly adherent to the serosa. Moreover, there appeared to be no attachment of the diaphragm proper to the anterolateral thoracic wall. The loops of intestine were freed from the margins of the sac and replaced in the abdominal cavity.

Fig. 14. X-ray after barium enema showing all the intestines below the diaphragm.

It was then apparent that the left lobe of the liver would have to be brought down before the main breach in the diaphragm could be repaired. A plane of cleavage was established between this lobe and the sac. This was extended until the liver lobe was entirely free. It was then replaced in the left side of the abdomen. The hernial sac was pulled down to the level of the musculature of the diaphragm and infolded with fine silk. The size and position of the apertures in the diaphragm are shown in fig. 13. In order to secure approxi-

mation of the edge of the diaphragm to the anterolateral wall, it was necessary to put some degree of tension on the diaphragm. Then with a sharp needle the suture of No. 12 silk was carried through the ribs along the arc which formed the anterior boundary of the open space, thus completely closing the thorax from the abdominal cavity.

This child made an uneventful convalescence. Gastrointestinal x-ray films on December 20, three weeks after operation, revealed all the intestines below the diaphragm (fig. 14). Two days later she was discharged recovered.

Two months later a report came from her physician in Baltimore. The child had improved rapidly. Her color was better and appetite excellent. Her father reported ten months later that she had grown three inches and gained fourteen pounds.

The evidence in this case indicates the retarding influence on mental and physical development of a congenital diaphragmatic hernia of this type. Moreover, it serves to show that the most intelligent form of medical treatment may be applied without success and that the period of arrested development may last for months or years. I believe that these patients tolerate surgical interference with comparative safety and should be operated upon after a reasonable trial of expectant treatment.

An opinion with which I find it difficult to agree is that expressed by Marquezy of Paris[7] in 1936. He reported a case of congenital hernia of the stomach in a child five years old who had been kept under observation for 3½ years after the diagnosis had been established. In spite of the fact that this child never willingly took her food, was anemic, underweight and difficult to manage, the author concluded that operation should not be done for five more years, believing that ten years was the age considered suitable for operation by most surgeons.

Orr and Neff[8] reporting successful surgical treatment in an infant 27 days old, collected sixteen cases in children less than a year old with a mortality of 47 per cent. Wider experience and a better knowledge of protecting or supporting infants while on the operating table, together with the most favorable form of approach and type of anesthesia, will undoubtedly reduce the present mortality.

In observations at five autopsies on infants with diaphragmatic hernia, I have found no adhesions between the herniated structures and the visceral and parietal pleura. The diaphragm at birth has only a slight upward convexity, rendering it more accessible from below. This is significant, because most of the hernias which have developed during embryonic life have occurred through the foramen of Bochdalek. Absence of adhesions and ease of access make lapa-

rotomy the more logical and safer approach. At birth the stomach is often found distended with gas, making reduction difficult from below. This air-bag can be collapsed promptly by passing a small rubber catheter into the stomach. As a rule, the small bowel is not distended, and if the abdominal incision is made parallel to the costal margin, it will be found less difficult to prevent evisceration of the replaced loops of intestine.

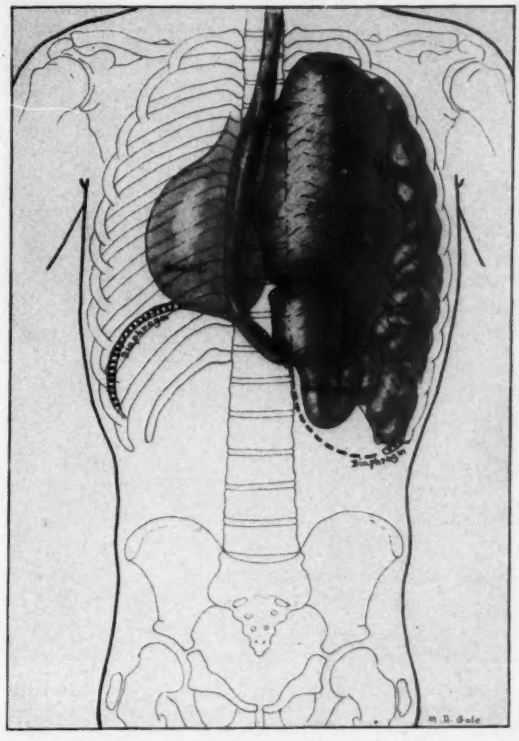

Fig. 15. Drawing made from x-ray film showing inverted stomach and loops of intestine in the left pleural cavity. Age of patient 2½ years.

Congenital diaphragmatic hernia in early infancy, therefore, is best treated by laparotomy at any time after the diagnosis has been established. Although Griffith,[9] Guy and Rand,[10] and other authors advocate delay of operation, I have experienced the misfortune of losing two infants on account of postponement. The first, six weeks old and acidotic on admission, died while we were attempting to secure fluid balance to combat dehydration as a measure preliminary to operation. The second, admitted at the age of 3½ months, anemic, dehydrated and undernourished, died four hours after op-

eration from shock. These deaths might have been avoided if both had been operated upon early by laparotomy.

There are two other cases of the congenital type which I wish to report briefly. The first is that of the girl who came to our hospital from Omaha when she was ten years old. At the age of 2½ years, her case was diagnosed as pulmonary tuberculosis. She was

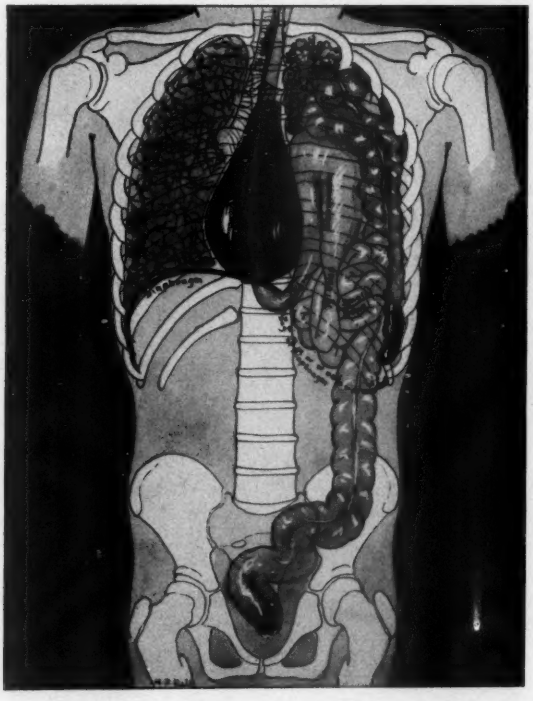

Fig. 16. Composite drawing from x-ray films showing herniated viscera.

sent to Denver, Colorado, for climatic treatment. When she was admitted to the Children's Hospital there, she was x-rayed. The large cavity observed on the x-ray film aroused suspicion of the presence of a hollow viscus in the chest, instead of cavitation associated with tuberculosis. A barium meal was given and the stomach revealed occupying the major portion of the left pleural cavity (fig. 15). Since her condition was precarious, operation was considered too hazardous.

She lived the life of a cripple until seen by Dr. Clarke and Dr. Brown of Omaha. They found her in a state of impending danger from attacks of obstruction associated with vomiting, convulsions, and cyanosis.

Figure 16, a composite drawing from x-ray films, shows the position of the organs when this patient was admitted to our Clinic. The stomach, small intestines, omentum, appendix, cecum, ascending and transverse colon, and spleen are in the left thoracic cage.

At operation by the thoracic route, we found the hollow viscera constricted at the aperture in the diaphragm, thus explaining the attacks of acute intestinal obstruction. The entire gastrointestinal tract with the exception of the descending colon occupied the left thoracic cage. The left lung was entirely collapsed. The left lobe of the liver also projected through the opening in the diaphragm. The spleen lay in the costophrenic angle. All the organs were replaced in the peritoneal cavity and the tear in the diaphragm sutured.

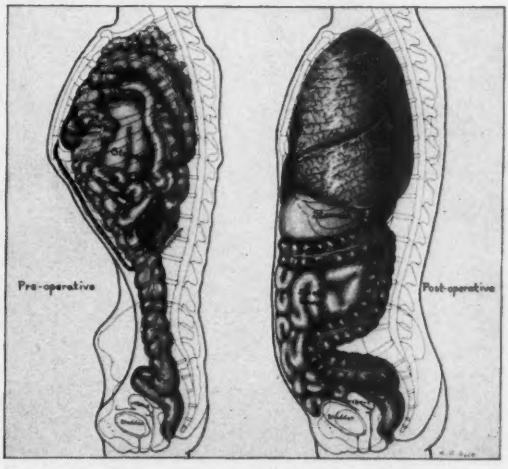

Fig. 17. Profile view of organs and contour of body before and after operation.

Figure 17 contrasts the position of the organs in thorax and abdomen and the contour of the body before and after operation.

This child weighed 46 pounds when she was admitted Feb. 18, 1935. Now, three and one-half years after operation, she weighs 120 pounds, enjoys hiking and swimming and leads a normal active life.

The third case in this series is that of a boy 13 years of age. From birth he had frequent spells of colic with severe attacks of vomiting which lasted several days at a time. From the age of 18 months attacks of abdominal pain and belching came on every two weeks. There was no cyanosis or dyspnea. When five years old he was thought to have whooping cough. X-ray examination at this time showed pneumothorax and collapsed left lung. A few weeks

later, x-ray examination with barium enema showed the colon in the left pleural cavity. Phrenicotomy had been done without improvement.

In spite of these handicaps the boy managed to lead a fairly active life. At our hospital physical examination showed the left chest somewhat dull over the entire middle and lower portions with breath sounds diminished over these areas. Intestinal râles and gurglings were audible throughout the left chest. There was a marked dextrocardia.

Fig. 18. Composite drawing from x-ray films showing herniated viscera.

X-ray examination April 2 showed the presence of small and large bowel in the left pleural cavity. Figure 18 shows the atonic, markedly dilated stomach in the abdomen and the intestines in the left thoracic cage.

This patient was operated upon by the transthoracic route. The chest was found to contain the major portion of the colon with cecum and appendix, the small intestine, and the spleen. The defect in the diaphragm extended laterally from the aorta to the periphery.

This large aperture accounted for the absence of intestinal obstruction.

After a somewhat protracted convalescence, this patient was discharged on June 15. The postoperative result is shown in fig. 19. On admission he weighed 63 pounds. He now weighs 118 pounds. He is five feet, eight inches tall, a junior in high school, and plays on the baseball team.

Fig. 19. Small intestines and colon in normal position below the diaphragm.

The next case is one of traumatic diaphragmatic hernia. D. S., a housewife, aged 42, was admitted May 18, 1936, with the complaint of gaseous distress and digestive disturbances. On March 8, 1935, she was injured in an automobile accident. With her were her two daughters. Both were killed instantly. Therefore the shock was extreme and lasting. The patient was removed to a hospital where an x-ray examination revealed the left lung collapsed and the heart displaced to the right. Apparently these findings aroused no suspicion of diaphragmatic hernia. During her stay of ten days she found breathing and deglutition difficult and could not lie on her left side. She complained of epigastric distress, more severe when she was in the recumbent posture.

In February, 1936, eleven months after the accident, while in Providence, R. I., she had an attack of grippe. Her physician, Dr. Walter Jones, after taking a careful history, examined the chest by auscultation and heard intestinal rumblings in the left pleural cavity. Immediately suspecting a diaphragmatic hernia, he had the patient x-rayed after a barium meal. This procedure proved his suspicions to be correct.

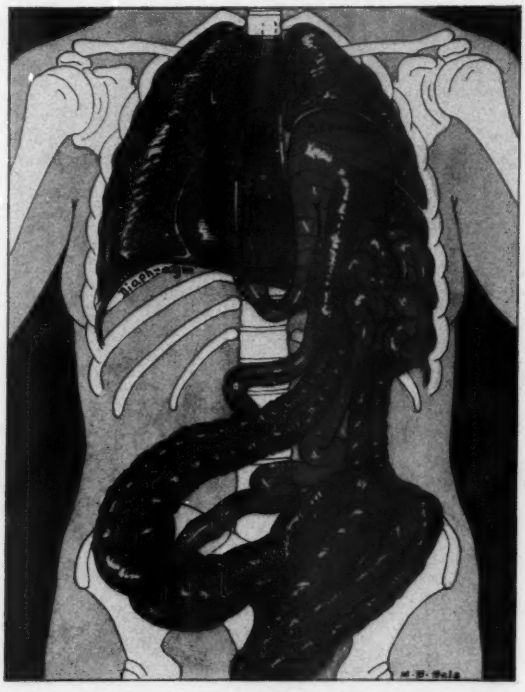

Fig. 20. Composite drawing from x-ray films showing herniated viscera.

When she came to our Clinic, physical examination elicited all the signs found by Dr. Jones, confirming his diagnosis.

Figure 20 shows the entire stomach, part of the small intestines, and splenic flexure of the colon in the left thoracic cage. The points of compression of the ascending and descending loops of bowel suggested that the torn leaf of the diaphragm was depressed by the hollow viscera above and lay in an almost horizontal line opposite the eleventh rib. The loops appeared to be free and there was no delay in the passage of barium throughout the entire colon. Figure 21 shows a lateral view. The diagnosis was left diaphragmatic hernia of traumatic origin.

Under gas-oxygen positive-pressure anesthesia, an incision was made over the seventh and eighth ribs on the left side and a segment of the eighth rib removed. The seventh rib was then cut posteriorly to obtain adequate exposure. The lung was seen partially collapsed by pressure from the stomach, transverse colon and small intestine. The spleen also was above the diaphragm adherent to the margins of the aperture. The left phrenic nerve was crushed with a hemostat. The herniated viscera were then replaced in the abdomen and the large tear in the diaphragm closed with a continuous running suture of No. 12 silk reinforced with several interrupted sutures of the same material.

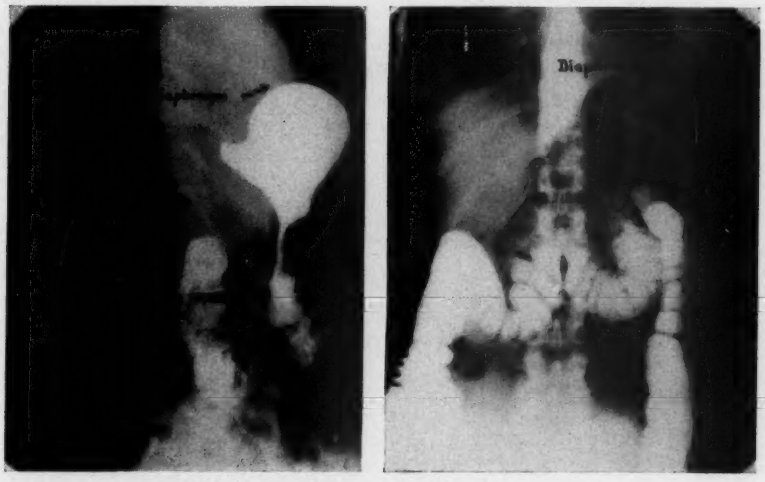

Fig. 21a. Lateral view showing the stomach in its normal position below the diaphragm. Fig. 21b. Anteroposterior view showing the colon entirely within the abdomen. The arrows point to the dome of the diaphragm.

The patient made an uneventful recovery. June 11, nineteen days after operation, x-ray examination showed all the viscera in their normal position below the diaphragm. Figure 21a shows the stomach and small intestines below the diaphragm. Figure 21b shows the barium enema outlining the colon which is below the diaphragm. The left leaf of the diaphragm is now at a much higher level.

This patient left the hospital three weeks after admission. She was in good physical condition, eating and sleeping well. Oct. 1, 1937, seventeen months later, she reported that she had remained well and had no complaints. X-ray examination again showed the abdominal viscera in normal position below the diaphragm.

Esophageal Hiatus Hernia

The following cases of hiatus hernia differ widely from congenital or traumatic hernia, but show little variation from one another. The first case is that of a man, 65 years of age, admitted Jan. 12, 1934, with the complaint of vomiting and loss of weight. Dr. A. R. Moses of Charlton, Mass., had established the diagnosis of hiatus hernia by x-ray examination. Since this patient had suffered no serious illness throughout his life and no lesions of the vascular system were demonstrable, he was considered a fairly good operative risk in spite of his age. For six months he suffered intermittent attacks of vomiting soon after eating. The vomitus had never contained blood.

On admission his appearance was that of a sick man. We considered him a poor operative risk and kept him on a medical régime for two weeks. During this time he improved noticeably, but we were still dubious concerning his ability to withstand surgical intervention and felt that the results would be extremely problematical. Dr. Moses, however, stated that, since he had reached the end of his resources medically, operation should be undertaken. Subsequent events justified his recommendation.

X-ray examination three days after admission revealed dextrocardia and an esophageal hiatus hernia with 7 cm. of the cardiac end of the stomach in a sac about the diaphragm.

January 25 operation was done by the thoracic route. This proved a most fortunate choice of approach, because upon opening the pleura we found definite evidence of an old diaphragmatic pleurisy in the form of a dense network of adhesions from the entire base of the left lung to the diaphragm (fig. 22). There were also multiple bands of adhesions between the hernial sac and the left lung. It was obvious that so extensive a field of adhesions could be dealt with in a safe manner only by a thoracic approach. The adhesions were severed across the dome of the diaphragm toward the hiatus through which the hernial sac protruded. To facilitate reduction and repair of the hernia, the left phrenic nerve was injected with alcohol.

The hernia was reduced, the sac removed, and the aperture in the hiatus repaired. The thoracotomy wound was then closed by interrupted sutures of silk worm gut.

Convalescence was retarded by an attack of bronchopneumonia during which we feared he would not recover. At the time of discharge, six weeks after operation, he was eating and sleeping well and had no complaints. Nine months later his physician reported as follows:

"Mr. B. reports that he has no complaints and feels better than he has felt for years."

November 6, 1936, two years and nine months after operation, he wrote again:

"This man is still in excellent health, eating all foods and living a very happy existence."

Fig. 22. A dense network of fibrous bands between the inferior surface of the lung and the superior surface of the diaphragm, entirely obscuring the esophageal hiatus hernia.

The second case of hiatus hernia is that of a woman, age 70 years, who stated that she had stomach trouble as long as she could remember. For eight years she had suffered from epigastric pain which radiated to the back and was frequently associated with vomiting. At first these attacks occurred from two to six months apart, but for six years substernal and epigastric distress had persisted. Her discomfort is best described in her own words, that "she was always putting one meal on top of the other."

A chronic cough with hoarseness and aphonia had been troublesome for years. Symptoms became more acute four months before

entry. She was x-rayed and from the roentgenogram it was thought that she had a cancer of the stomach. She was sent to a cancer hospital at Pondville, Mass. Here they were unable to corroborate the diagnosis of carcinoma, but found that she had an esophageal hiatus hernia.

She was admitted to this hospital for operation January 18. Our x-ray examination confirmed the diagnosis of hiatus hernia. The hernia was repaired by a transthoracic approach. She made an uneventful recovery and left the hospital on February 10, twenty-three days after operation.

This patient returned February 26 for x-ray examination, which revealed the cardiac end of the stomach in normal position below the diaphragm. This woman has been completely relieved of gastric symptoms.

The third patient in this group is a woman, age 61 years, who had had diabetes for five years. Epigastric pain and distress had begun three years previously, after a fall. The onset of symptoms was gradual. Eructations and cramplike pain over the precordium became almost constant, and did not respond to medical treatment. Pain invariably followed deglutition. It was often spasmodic and radiated to the left shoulder. The pain often occurred at night.

In July, 1936, she went to a metropolitan hospital where an electrocardiographic tracing was interpreted as significant of coronary sclerosis. A cholecystogram was negative. While there she was placed on a diabetic régime and given insulin. She was sent home with advice to continue medical treatment and rest. No improvement followed and by October the epiphrenal syndrome became intolerable. She was x-rayed again and an esophageal hiatus hernia observed (fig. 23). She was advised to enter the hospital.

Our problem was essentially one of diagnosis. Inasmuch as coronary sclerosis was a factor to be considered, it was of the utmost importance to determine as accurately as possible the true cause of this patient's symptoms. Peptic ulcer, cholecystitis, and malignancy had been ruled out. The clinical picture did not have the appearance of a psychoneurosis. She was five feet, two inches in height, and weighed 167 pounds, hence, of the *pykniker* type, so frequently alluded to by German authors to describe the broad sterno-vertebral diameter.

This narrowed the diagnosis to lesions of the heart and cardiac portion of the stomach. An electrocardiographic tracing showed inversion of the T-wave in Leads 1 and 2. The R S T segment was slightly depressed in Lead 1. In Lead 4 there was absence of the initial downward deflection, although the T wave was in normal

direction. The tracing indicated coronary disease with suspicion of an old anterior infarct; there was also left axis deviation. Therefore coronary disease could not be excluded and there was also definite indication of trouble at the cardia.

We kept this patient under observation for eighteen days. Treatment consisted mainly of rest in bed. During this period she had many attacks of precordial pain, with numbness of arms, choking

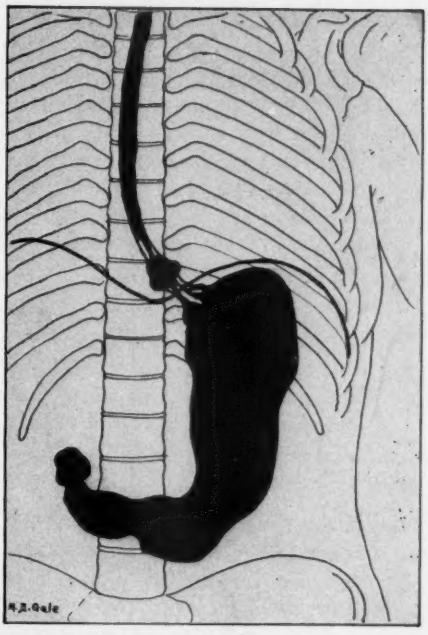

Fig. 23. Schematic drawing showing appearance of stomach by x-ray after barium meal.

sensation, inability to speak; the pulse became weak and irregular, rate 68. These attacks were relieved by vomiting, by eructation of gas, and by the insertion of a Levin tube.

Inasmuch as medical treatment brought no permanent relief, but the insertion of the Levin tube and eructation of gas did make her comfortable, we reached the conclusion that the predominating symptoms could probably be accounted for by insufficiency at the cardia.

Since the laboratory findings of the upper abdomen were negative, we decided to approach the cardia by laparotomy and examine carefully those organs which might present some pathology. The appendix, gallbladder, stomach and spleen were normal. There was no

enlargement of the pancreas. At the cardiac end of the stomach there was relaxation of the left crus. The hiatus admitted two fingers, but the condition could not be termed a true hernia.

In view of these unsatisfactory findings at the hiatus, and knowing that relief of symptoms was obtained with the indwelling Levin tube, it seemed probable that if the left leaf of the diaphragm were put at rest, the patient might find relief. So the abdomen was closed and a phrenicotomy done through an incision above the left clavicle.

This patient has had only one attack of pain since the phrenic nerve was severed. This attack occurred February 10, seventeen days after operation. Otherwise convalescence was uneventful. The following day a second electrocardiographic tracing showed Leads 1 and 2 as in the first tracing. In Lead 4 the Q R S deflection was downward. The T wave was not distinct, but tended to be slightly elevated. The diagnosis was still coronary disease, old anterior infarct, and left axis deviation.

It is now one year since this patient was operated on. She came to report that she has been entirely free from the spasmodic precordial pain, the left shoulder pain and the night attacks. Food still seems to lag before entering the stomach, but does not interfere with her doing regular household duties. What significance this may have on cardiac pain from intrinsic pathology can be determined only on further investigation.

The fourth case in this group was that of a female, aged 52 years. She suffered from dysphagia for eight years. Two years before admission she experienced a sudden inability to swallow food, water, or saliva. This dysfunction lasted for ten hours. Following this she had occasional attacks when she could not swallow coarse or bulky food. She was referred to the Eye and Ear Infirmary in Boston where a diagnosis of mediastinal tumor was made. X-ray examination revealed an esophageal hiatus hernia with about 7 cm. of the cardiac end of the stomach above the diaphragm. For three months before admission she suffered constant distress. She swallowed very slowly and with great difficulty. She suffered from dizziness, constipation, nausea, and regurgitation.

X-ray examination at our clinic confirmed the diagnosis of hiatus hernia. It was noted that the esophagus was tortuous, with the lower end about 5 cm. above the diaphragm. The upper loculus of the stomach when filled measured 7 cm. in diameter.

Operation was done by the abdominal route. The hiatus admitted four or five fingers. The adherent stomach was released and replaced in the abdomen and the hiatus closed snugly about the

esophagus. After operation there was still some difficulty in swallowing. April 8 x-ray examination revealed the stomach in normal position. There was an area of constriction in the esophagus at the level of the aortic arch. The esophagus was therefore dilated with a bougie. She returned for dilatation of the esophagus May 14, July 23, and October 29, 1936, and in June, 1937.

A letter received from this patient March 3, 1938, stated the following:

"It is just three years ago that I was operated upon. Before operation I could eat only soft foods and liquids and even these often caused choking. At times my throat felt completely blocked. Now I eat anything I like and have a wonderful appetite.

"I used to have dizzy spells and fall to the floor. Now I never feel dizzy. I used to feel suffocated when I lay down at night. Now I just go to bed and sleep. I am able to do all my housework and can even go out to parties and dance. Do you wonder that it seems like a miracle to me?"

Perhaps it is not in good taste to publish this letter in full. But there has been a good deal of unfavorable criticism about undertaking any form of surgical treatment to relieve patients suffering from hiatus hernia. It is of the first importance to exclude all other possible causes of symptoms. We agree that medical treatment should be given a fair trial but when that fails, it is our conviction that repair of the hiatus by laparotomy or thoracotomy is indicated.

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THE INTERPOSITION OPERATION FOR PROLAPSUS UTERI

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PROLAPSUS uteri is descensus of the uterus below its normal level in the pelvis. The extent of a prolapse varies from a slight descensus of the uterus to its complete escape through the vulvo-vaginal orifice. So long as the uterus remains within the vagina, the displacement is spoken of as an incomplete prolapse or prolapse of the first and second degree, and when the uterus has escaped through the vaginal ring it is a total prolapse or prolapse of the third degree.

The majority of cases of prolapse of the uterus are seen in women who have borne children, the condition being the result of damage caused by the passage of the child through the pelvis. Prolapse usually is accompanied with cystocele and rectocele.

Prolapse in the unmarried woman is not common. When seen it is usually the result of faulty innervation to the structures of the pelvis. When abnormal intra-abdominal pressure is exerted upon the uterus there is a resultant hernia or prolapse, and this is the most difficult type in which to get a complete cure. Much has been said and written about this type of prolapse, classifying it along with cases of spina bifida occulta.

The lacerated pelvic floor is not the cause of the prolapse. There are many cases of lacerated perineum of long standing in which the uterus is well up in the pelvis and in good position. However, it is natural to suppose that lacerations of the perineum have a great part to play in most cases, for the perineal floor acts as a support to the pelvic contents and when it has been damaged, with damage of the structures above the levator ani group, the uterus and other organs will certainly give way and come to the vaginal outlet. The next layer above the levator muscles is the endopelvic layer of the pelvic fascia, the main supporting fascia of the uterus and bladder. On the cadaver one can easily demonstrate the pelvic fascia as a direct continuation of the fascia of the abdominal wall, placed between the muscles and peritoneum. The relative position is identical and the visceral layer passes inward from the pelvic walls upon the upper surface of the levator ani muscle and forms a fascial diaphragm which closes the pelvic outlet. This is often called the pelvic diaphragm. It arises from the symphysis and pubic ramus: from the symphysis at about the level of the junction of the lower and

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middle thirds. It passes under the bladder and forms the anterior ligaments of the bladder. It surrounds the urethra and is intimately connected with the base of the bladder. It sends numerous fibers up along the walls of the bladder as well as downward along the urethra. The fascia passing under the base of the bladder is closely connected with the anterior wall of the supravaginal cervix and it helps to form the anterior vaginal fornix. This part of the fascia is very plain and is often called the "anterior true ligament of the bladder." It holds the bladder and urethra close to the symphysis.

The next part of the pelvic fascia arises from the pubic ramus and the white line, first at the level of the top of the obturator foramen and from the inner surface of the spine of the ischium. It runs inward above the levator ani muscles toward the vagina, bladder, cervix and rectum, and divides into the vesical, vesicovaginal, rectovaginal and rectal layers. The posterior part of the fascia here surrounds the rectum and follows its walls as the rectal layer, giving some support to the upper part of the posterior vaginal wall where it is in relation to the anterior wall of the rectum. This part constitutes the recto-vaginal layer. The anterior position of this fascia is stronger and thicker than the rectal portion and constitutes the vesicovaginal layer. As it runs inward toward the viscera it divides into numerous layers and becomes intermixed with loose connective tissue which makes the demonstration of the whole fascia almost impossible, close to the viscera. Nevertheless, the continuity can be shown and the different layers followed, to lose themselves upon the walls of the viscera or to join the fascia of the other side. It is connected to the sides of the bladder and continuous with the anterior fibers of the fascia which pass under the base of the bladder. It passes across the pelvis between the anterior vaginal wall and the base of the bladder and is attached to the cervix uteri and the lateral vaginal fornices, above which it passes to reach the cervix and to become continuous with the rectovaginal layer between the posterior vaginal fornix and the rectum below the posterior culdesac peritoneum.

Of the ligaments of the uterus the sacro-uterine are most concerned in maintaining the position of the uterus and the round and broad ligaments play very little part in supporting it. Retrodisplacement of the uterus is claimed by some to be a predisposing cause in prolapse. This I do not believe to be the case. But after a separation of the pelvic diaphragm when the uterus begins to descend, we have a retrodisplacement which is really the beginning stage of prolapse. It is a concomitant and not a predisposing cause, although it is a development in all cases.

Prolapse of the uterus including cystocele is of course a very old subject. In fact the condition was so distressing and caused women

so much incapacitation that it was one of the first human ailments to be recorded and studied. Far back in medical history one finds reference to uterine prolapse, and the treatment offered for its correction through the years constitutes a most interesting history. However, as the years passed by the treatment has been improved and today the operative treatment for uterine prolapse is a safe and well established procedure.

In studying prolapse of the uterus it is well to consider cystocele as the two conditions are inseparable. Cystocele is hernia of the bladder through the vesicovaginal septum and is generally the result of injury at childbirth with sub-involution. The protrusion of the bladder and the senile changes in the tissues which occur after the menopause gradually increase the size of the hernia. The hernial opening in extensive cases of cystocele extends sagitally from the pubes to the cervix and transversely across the entire anterior portion of the pelvis. The anterior vaginal wall is usually so much thinned by stretching and laceration that no definite borders of the hernial opening can be palpated. A urethrocele with thickening of the mucous membrane over the body of the urethra is often coexistent.

Uterine prolapse is a hernia of the uterus. In prolapse of the uterus the broad and ureterosacral ligaments are elongated, the vaginal canal dilated, the perineum relaxed and usually lacerated. The cervix and the body of the uterus are frequently enlarged from passive congestion, edema and hyperplasia. The cervix may be cystic and eroded as a result of laceration, infection and friction. To cure the cystocele it is important to close the hernial opening through which the bladder protrudes and the best way to close this opening is to interpose either the uterus or its ligaments.

It is interesting to note that as far back as 1871 operations were described for the correction of cystocele and from that date to the present time numerous operations have been described but none has offered much improvement over those of the pioneers in this field.

It must be remembered that whatever type of operation is chosen for prolapse of the uterus, the accompanying cystocele must be considered. To cure the prolapse one must select an operation that will cure the cystocele which is the most troublesome part of the prolapsed organs. Also, repair of the pelvic floor is indicated in 100 per cent of all cases. Many cases of prolapse of the uterus have an elongated cervix which should be amputated.

Intelligently to discuss the treatment for prolapse of the uterus with its accompanying cystocele we must first determine whether childbearing is, or is not, a factor to be considered. The problem for consideration in the young childbearing woman is absolutely dif-

ferent from that in a woman whose uterus is incapable of reproduction or is past that period. The methods at our disposal for the correction of prolapse when childbearing is not a factor, are more certain of a permanent cure in their final results than are the methods available where the parts must be called upon to bear the strain of subsequent labors.

The treatment for prolapse of the uterus is operative and non-operative. There are many cases of prolapse occurring in the aged which for constitutional reasons are not suitable for operative work, even though the work is vaginal and usually carried out under local anaesthesia. These cases can best be treated by pessary. The Menge pessary is by far the most satisfactory. It is made in several sizes and consists of a hard rubber ring with crossbar in one piece, and knob or handle in another piece. The ring is first introduced after replacing the uterus in the pelvis and then the knob is fastened into the ring by a slot in the cross bar. This knob or rudder keeps the ring in place and does not allow it to turn over and fall out. The good qualities of this pessary are only appreciated after one has used it for some time.

The operative treatment for prolapse of the uterus must be one that will suit both the childbearing and the non-childbearing woman.

The Childbearing Woman

1. Abdominal and vaginal operation.
   a. Abdominal: shorten the round and sacro-uterine ligaments.
   b. Vaginal: repair cystocele and rectocele.
2. Abdominal: hysterectomy, complete or supravaginal.
   Repair cystocele and rectocele.
3. Vaginal: plicating the broad ligaments across the anterior surface of the uterus. Amputate the cervix. Repair rectocele.
4. Vaginal: hysterectomy and interposition of the round ligaments (Mayo). Repair cystocele.

The Non-Childbearing Woman

1. Abdominal and vaginal operation.
   a. Abdominal: fixing the uterus to the abdominal wall.
   b. Vaginal: repair cystocele and rectocele.
2. Vaginal: plicating the broad ligaments across the anterior surface of the uterus. Amputate the cervix (Baldwin: Fothergill). Repair rectocele.
3. Vaginal: Interposition operation (Watkins). Repair rectocele.
4. Vaginal: hysterectomy with interposition of the round ligaments. (Mayo). Repair rectocele.

In the interposition operation, the bladder is separated from the vaginal wall and the uterus, the anterior peritoneal pouch is opened, the corpus uteri is brought down under the raised bladder, and is

fastened there. The excess of vaginal wall is trimmed off and the vaginal wound closed. Then the pelvic floor is repaired as usual.

By this operative procedure, the corpus uteri is interposed between the bladder and the anterior vaginal wall and is securely fastened in a position to cover effectively the weak place (vaginal opening) in the pelvic floor and give permanent support under the atonic base of the bladder. The operation is here described again. Illustrations showing the details will be found in Crossen's textbook, *Operative Gynecology* (St. Louis: C. V. Mosby Co., 1938).

1. *Separation of vaginal wall from bladder.* Separation of the vaginal wall is begun by making a median incision extending from just below the urethra to the cervix. The incision passes through the vaginal wall proper and the underlying attached fascia. There are two planes of cleavage: one, not very apparent in the median line, between the vaginal wall and the fascia, and the other more evident, between the fascia and the bladder. The separation should take place along the latter plane, so that the fascia remains attached to the vaginal wall and thus give a stronger support under the interposed uterus. The cleavage plane between the fascia and bladder wall is most easily identified in the posterior part of the incision, consequently it is well to begin the separation there.

2. *Separation of bladder from uterus.* Laterally the bladder is easily pushed off the cervix, but in the median line it is usually held by some connective tissue fibers which must be divided with scissors or knife. The bladder is then pushed off the uterus by gauze dissection up to the vesico-uterine fold. The lateral dissection of the bladder from the broad ligaments should be only sufficient to give room for bringing down the fundus uteri.

3. *Opening of vesicouterine peritoneal fold.* Difficulty is often experienced in identifying this fold. It may be recognized in some cases by a slight difference in color. Usually, however, it must be recognized by touch. After the fold is located it is caught with a forceps and opened with the scissors.

4. *Bringing out corpus uteri.* The corpus uteri is brought out by tenaculum forceps, placed one above the other. The delivery of the fundus is facilitated by pushing the cervix back in the pelvis.

5. *Suturing vesical peritoneum to back of uterus.* The edge of the vesical peritoneal flap is sutured across the posterior surface of the uterus at about the level of the internal os. This practically shuts off the peritoneal cavity.

6. *Suturing fundus uteri to fixed tissues near pubic arch.* The uterus is to be fastened in its new location between the bladder and the vaginal wall. With operators generally, the simple sewing over of the vaginal wall was depended on to hold the corpus uteri in position. This gave a good result in many cases, but there were some recurrences. In order to prevent recurrences, with sutures the fundus is fastened to firm tissue under the pubic arch which prevents both downward and upward displacement.

7. *Closure of vaginal incision.* The excess of vaginal wall is trimmed off. The reduced flaps are then sutured together in the median line so as to form a good side-to-side sling for the corpus uteri to rest upon. The sutures closing the vaginal wound should take hold of the underlying uterus. These sutures are entirely buried when the vaginal flaps are brought over. They fasten the fundus uteri to tissues that resist both downward and upward displacement.

8. *Repair the pelvic floor.* A thorough repair of the relaxed pelvic floor is of course a necessary part of the operation.

In our series of several hundred cases of prolapse of the uterus with cystocele operated upon at Barnes hospital, the Watkins interposition operation was performed in about 60 per cent with very few failures. Those few cases that were failures were due to an error in judgment in selecting the type of operation for the particular case. In most instances the uterus that was interposed was either too large or too small or it protruded completely out of the vagina rendering the case unsuitable for an interposition operation. However, in every case the patient was finally cured by later removing the uterus and interposing the round ligaments. The other 40 per cent of our cases had one or the other type of operation performed as listed in the above classification. The second and third most frequent type of operations performed by us were vaginal hysterectomy with interposition of the round ligaments (Mayo), and the Manchester or Fothergill operation. Whenever possible and the operation could be carried out we performed the interposition operation (Watkins).

In selecting a case for the interposition operation, one must bear in mind that it is employed only in non-childbearing women and the prolapse is not beyond the second degree. The uterus is neither too large nor too small and the cervix is not pathologic. Always precede the interposition operation with a gentle curettage of the uterus to rule out fundus malignancy and always repair the pelvic floor.

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THE OPERATIVE TREATMENT OF VESICOVAGINAL AND RELATED FISTULAS

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IN the development of the successful operative treatment of vesicovaginal fistulas we must turn to the South, and to Alabama. It was in Montgomery that J. Marion Sims, after many trials and as many failures, finally solved this age-old surgical problem. He had devised instruments and a position for the adequate exposure of the fistulous tract, a technic to denude and suture the margins of the opening and a catheter for postoperative drainage of the bladder, yet complete union did not occur. Finally in his thirteenth attempt to close the fistula of Anarcha, one of his negro patients, he used four interrupted sutures of fine silver wire which he had had made especially for this purpose. Whereas, after his previous operations in which silk sutures were used, a cystitis with swollen urethra, and urine loaded with thick ropy pus, had always followed, this time with the silver wire sutures the urine remained clear and limpid, there was no inflammation and a successful closure followed. This was in 1849.

Sims knew that another remarkable surgeon for his time, Dr. J. P. Mettauer of Virginia, had previously closed a vesicovaginal fistula with sutures of lead wire, and he had tried this material, but with failure. Mettauer reported six cases of a vesicovaginal fistula for which he had used sutures of lead wire. In one there was a primary successful closure, in a second a troublesome secondary fistula formed in the tract of the wire which eventually closed under cauterization. Failure in the other four patients is suggested, as the author does not mention the results of his operations. Mettauer whose instruments and wire may be seen in the Mutter Museum in Philadelphia is to be credited with the first successful closure of a vesicovaginal fistula and for the introduction of a metallic suture for this purpose. A metallic suture in the form of gilt wire was used by Gosset of London, in 1839, who likewise employed a knee-chest position and perineal retractor, but apparently the result obtained did not encourage the continued use of the material. Sims, using a finer, better metallic suture, developed a technic so successful that he is said to have cured over 200 patients of vesicovaginal fistula.

When we consider the many failures that follow present day operations for vesicovaginal fistula, we marvel that nearly ninety years ago Marion Sims, without the advantages of asepsis, trained

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assistants, nurses or modern methods of illumination, probably closed more vesicovaginal fistulas than any surgeon who has followed him. Doubtless the operation may now be done with more uniform success and with less danger, but that it is not may be attributed to the fact that many ignore the principles laid down by Sims. He operated entirely through the vagina; yet at present with greater danger and often without success operations for vesicovaginal fistula are unnecessarily undertaken through the abdomen or through the bladder. I have encountered no patient with a fistula of bladder that could not be closed through the vagina, although some of these patients had previously had unsuccessful transabdominal operations.

Catgut has largely replaced the much more effective silver wire for sutures. Catgut is convenient but induces a local inflammatory and degenerative reaction inimical to prompt and sound healing. Now we have an even less irritating, stronger and more pliable metallic suture than silver wire in annealed rustless steel wire. Not only have Sims' fine wire sutures been ignored but also his silver sigmoid catheter, and patients after the operation are put to the discomfort of lying upon their face for a week or more, or even of having a catheter passed through the tissues lying between the symphysis and urethra. After the fistula is properly sutured I have found little need for restriction of the patient's position in bed.

Occasionally a part or all of the vagina is closed off (colpocleisis) as used by Simon and others before the success of Sims' technic was appreciated. The disadvantages and dangers of adding such a diverticulum to the bladder are to be emphasized. A bladder which on closure has only a capacity of 15 to 30 c.c. will usually dilate rapidly after the fistula has been closed.

A fistula with viable margins, well denuded, adequately mobilized and closed in layers with very fine, non-irritating wire and protected from tension by continuous catheterization, usually heals within a few days. After the operation the patient may be placed in a comfortable position: ordinary movements in bed rarely do any harm. The patient may be permitted to be out of bed in from five to ten days and to leave the hospital in from eight to fourteen days. If there is much avascular cicatricial tissue, or if the urethra has been extensively destroyed or the parts heavily irradiated by radium or roentgen rays, healing, of course, may be delayed, as in the following patient:

Mrs. L., aged 43, had undergone amputation of the cervix and, in the fear of carcinoma, prolonged application of radium had been made. Later marked vesical symptoms developed and what appeared to be a new growth on the base of the bladder was treated by fulguration.

When first seen she was constantly crying out from the intense pain of a violent cystitis and had been taking opiates for several months. On vaginal examination a thickened, indurated vesicovaginal partition was palpated. Was this due to the spread of a carcinoma or to the reaction to the application of radium and the electrical treatment? To determine this and also to place the bladder at rest a free opening was made through the vesicovaginal partition. Sections of semi-necrotic tissue were taken for microscopic study; there was no evidence of malignancy. The vaginal cystostomy gave the patient relief from the pain and the severely burned tissues had recovered sufficiently in six months' time so that the vesicovaginal fistula was then successfully closed with the alloy wire sutures without difficulty and to the complete relief of the patient.

Before discussing the technic of closing the fistula, it is desirable to consider common conditions leading to failure.

1. *Infection.* Active cystitis, vaginitis, irradiation burn, or intense erythema of the surrounding skin should be relieved by douches, topical applications, drying powders and exposure to the air before operation is attempted. Irrigations with boric solution or, for a more intense infection, a 1 to 5000 nitrate of silver or in certain cases the carefully supervised administration of sulfanilamide is valuable. Ointments should not be used nor should solutions of lysol or those containing mercurials in sufficient concentration to be irritating to the bladder. The irritated surrounding skin usually heals rapidly under repeated irrigation with boric solution, diluted aluminum acetate solution or lead water, followed by drying and dusting with zinc stearate powder. For tuberculous lesions I employ a roborant general regime together with tuberculin injected subcutaneously every 4 to 7 days over the deltoid muscle. Rapidly increasing doses are used until a sharp febrile reaction is produced, after which the injections are continued weekly in amounts sufficient to produce a distinct but not a violent reaction. Nitrate of silver locally is contraindicated in tuberculosis.

2. *Inadequate mobilization and denudation* commonly results from inadequate exposure of the fistula. Usually I employ the lithotomy position, but the knee-chest or Sims' position may be desirable in special cases. As a rule I use a Simon or a weighted posterior vaginal speculum together with a narrow trowel and at times a lateral vaginal retractor. Often a combination of the lithotomy and the Trendelenburg position is of advantage. If the introitus is narrow, an episiotomy or rarely even a Shuchardt incision may be necessary for adequate access. I have not found occasion to use the latter. Commonly the margins of the fistula are fixed and rather high. The edges of the fistula should be split or pared and the line of cleavage between the vaginal wall and the bladder located and followed sufficiently to mobilize freely the vesical wall. The possible close proximity of one or both ureters should always be con-

sidered, especially if the fistula is large or high in the vaginal vault. Often it is desirable to introduce ureteral catheters before beginning the dissection, as illustrated by the following:

Mrs. C. was referred after three previous unsuccessful operations. The fistula was a large one and in liberating the edges of the bladder both ureters were entered. The cut ends of the ureters were freed and carried into the bladder between separate interrupted sutures of very fine alloy steel wire, over which the denuded vaginal margins were carefully united also with the wire. A small soft rubber mushroom catheter was introduced through the urethra.

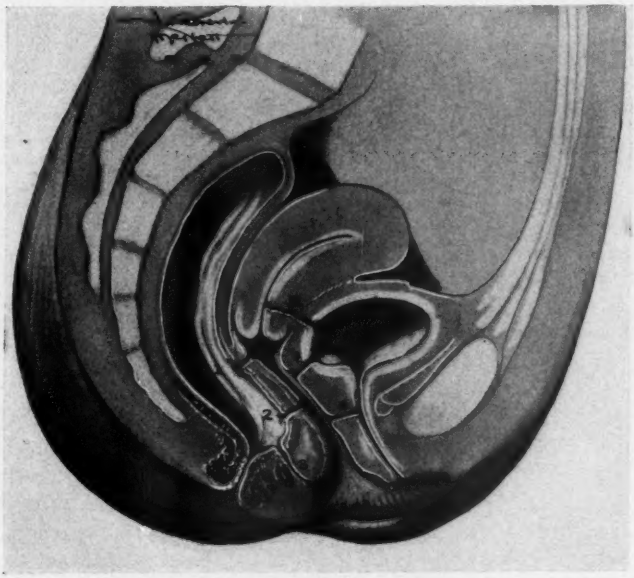

Fig. 1. 1, 2, 3. Rectovaginal fistulas. For the successful closure of types 1 and 2, a temporary division of the rectovaginal septum is occasionally necessary. For those entering the fourchette (3) simple division of the overlying tissues, without suture, usually gives relief. A. Vesico-uterine fistula to be treated by separating the bladder from the uterus through an anterior vaginal incision. B. The usual site of vesicovaginal fistula. C. Urethro-vaginal fistula; the overlying tissues being thin, additional covering is to be mobilized from the sides of the urethra and united.

There was no leakage, the catheter was removed about the eighth day and the patient discharged about 19 days after operation without untoward symptoms. Had ureteral catheters been introduced before the operation, the fact that the ureters ran very close to the margins of the fistula would have been apparent.

If the fistula is high the bladder is separated from the anterior face of the uterus to, or if necessary even through, the peritoneal reflection. In this way the part of the bladder involved by a high,

or a vesico-uterine fistula may be mobilized and brought into the vagina for suture as in the following case:

Mrs. Loretta C., age 33, 4 para, housewife, was admitted to the Temple University Hospital, March 16, 1931 for vesicovaginal fistula. On Nov. 20, 1929, a vaginal hysterectomy and plastic operations had been done for lacerations of childbirth and vaginal bleeding. The operation was followed by the constant leakage of urine. In March, 1931, the opening in the bladder was sutured without success. In June, 1931, another surgeon operated without controlling the leakage.

Laboratory studies showed pyuria, anemia marked by 40 per cent hemoglobin and 2,830,000 red blood cells with some anisocytosis and poikilocytosis. Under spinal anesthesia with procaine a 2 cm. opening from the vaginal vault into the bladder was encircled by an incision and the bladder separated from the anterior vaginal wall opening the peritoneal cavity. The margins of the opening into the bladder were inverted and united by interrupted sutures of fine silver wire and the line of union transplanted behind the anterior vaginal wall with mattress sutures of 00 catgut with which the vaginal opening was also closed. A mushroom catheter was inserted in the bladder and a light gauze packing of iodoform gauze into the vagina. There was no secondary leakage or untoward symptom. The catheter was removed eight days after operation, and two days later the patient was permitted out of bed. She was discharged thirteen days after the operation as cured.

For sutures and ligatures I now employ only very fine (35 to 38 B and S gauge) annealed alloy steel wire. This soft wire made of chromium, nickel and steel is non-irritating and may be buried permanently in the tissues even in a septic field. It is much stronger and more pliable than silver wire and should be tied in a square or surgeon's knot and the ends cut very short. While it is desirable not to leave the wire exposed on the mucous surface of the bladder, there is less tendency for urinary incrustation than upon most other permanent suture materials. In the case of a large enterovesical fistula where a recurrent cancer of the colon had ulcerated into the bladder with fecal contamination, the posterior wall of the bladder as well as the involved bowel was resected and closed with the alloy steel wire. There was no urinary leakage and a year later the wire exposed on the mucous surface of the bladder was bright and free from incrustation. However, it is wise not to leave the wire exposed to the urine in the bladder even though calcareous incrustation seems to occur rarely.

I prefer a very fine, full-curved needle such as is used by opthalmologists. Preferably two rows of inverting, interrupted wire sutures should be introduced into the bladder wall after which it is wise to fill the bladder with saline solution to make sure that the wound is water tight. The ends of the outer row of interrupted vesical sutures may be brought through the vaginal wall to one side of the vaginal opening and tied on the vaginal side, leaving the

ends long enough to facilitate later removal. In this way the lines of incision in the bladder and vagina are staggered, insuring more secure closure. Finally the vaginal wound is closed, preferably with interrupted 35 or 32 rustless steel wire. If the perineum has been incised this also is united with layer interrupted buried and superficial sutures of fine wire. The buried wire rarely gives trouble and need not be removed. With thick, well-united deep vesical layers, we often unite the vaginal edges with a continuous submucous suture of fine (o or oo) plain catgut to obviate the difficulty of removing the wire sutures from within the vagina. However, only the fine wire should be used to ligate vessels and no catgut placed in the bladder wall or deep layers. In removing wire sutures

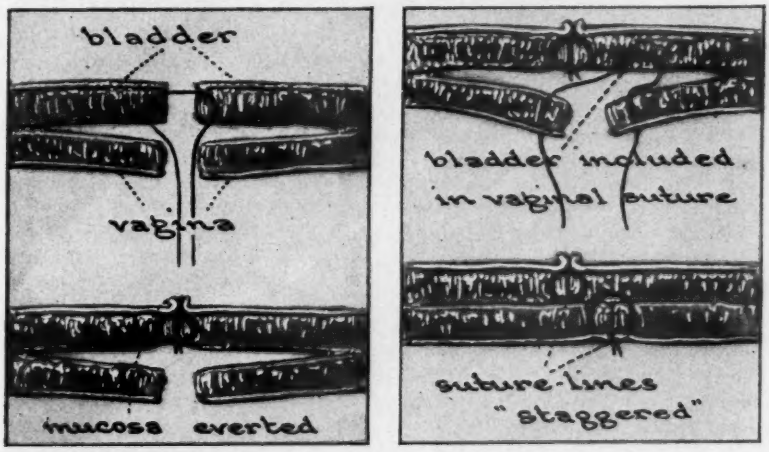

Fig. 2. Illustrating the method of mobilizing the bladder and bringing it through the vaginal incision for suture of the higher vesicovaginal or vesico-uterine fistulas.

from the vagina the Sims or knee-chest position and a very narrow Sims or Simon speculum are of great aid.

Continuous postoperative catheterization is desirable to keep the bladder at rest and to avoid tension on the line of suture. While an indwelling catheter of rubber produces more reaction than one of silver, there is a convenience in using a small soft rubber mushroom catheter. With a very small bladder or a sutured urethra such a catheter may, however, by pressure lead to secondary leakage as in the following case:

Mrs. D. had a large vesicovaginal fistula which involved also the proximal urethra and neck of the bladder. Several unsuccessful operations had been done and the bladder was hardly the size of an English walnut. Closure was made with alloy wire and a mushroom rubber catheter introduced. Leakage of urine

was present the third day after operation and the mushroom end of the catheter was found protruding between sutures at the neck of the bladder. Resuture and the substitution of a different type of catheter was followed by primary water tight union and by rapid increase in the capacity of the bladder. In such a case the small silver sigmoid catheter devised by Sims is very useful. To avoid harmful traction it should not be attached to a drainage tube.

Combined vesicovaginal and vesico-enteric fistula, especially if the small intestine is involved, may present serious and difficult operative problems. The escape of the very erosive small intestinal contents in association with the urine often leads to intense irritation, ulceration and destruction of pelvic tissues including the vagina, external genitals and surrounding skin. Not only is the patient in constant distress from the irritating discharges but exsanguinating hemorrhages may follow. The early closure of the opening in the small bowel is of paramount importance and may necessitate an abdominal section, as illustrated in the following case:

Mrs. L., a frail, married woman of 28 years, was admitted for a radical operation after a colostomy had been done elsewhere for an advanced and supposedly inoperable carcinoma of the rectosigmoid. A one stage abdominoperineal combined proctosigmoidectomy, panhysterectomy, resection of the left ureter, invasion of the base of the bladder and resection of the upper half of the vagina was performed, which was followed by leakage through the pelvis from the bladder and anastomosed left ureter. At a second stage suture of bladder from below, an adjacent prolapsed and adherent loop of small bowel was entered and sutured. The sutured bowel partly broke down with secondary tormenting erosion of pelvic tissues from the intestinal leakage. The involved intestine was therefore delivered through an abdominal incision, the fistulous portion resected and sutured with fine alloy steel wire and the abdominal incision also closed with buried alloy steel wire sutures. The patient was able to return home in about one week where she has gained weight and rapidly improved.

I have had a number of experiences with urinary or large intestinal fistulas associated with fistula of the small bowel—a most distressing and dangerous combination. The small intestinal leakage is not only exhausting but may erode vessels with even fatal hemorrhage. Our experience has taught us to separate and close the open small bowel early through an abdominal incision, leaving this sutured loop of bowel well separated from any other fistula.

Rectovaginal fistulas may be divided into those lying above and those lying through or below the anal sphincters, and also into those the result of trauma and those produced by disease (malignancy, fistula in ano, tuberculosis, syphilis), traumatism or irradiation.

The lower fistulas lying below the upper level of the internal sphincters and opening into the fourchette or lower vagina lead to the entrance of fecal matter into the vagina and vaginitis. Under

local anesthesia or gas-oxygen sufficient relief may usually be obtained by passing a probe or grooved director through the tract and dividing the overlying tissues in the midline. After this simple procedure which usually does not require confinement in bed fecal matter no longer enters the vagina and after cicatrization the patient as a rule has sufficient sphincter control to consider operative closure of anal ring unnecessary.

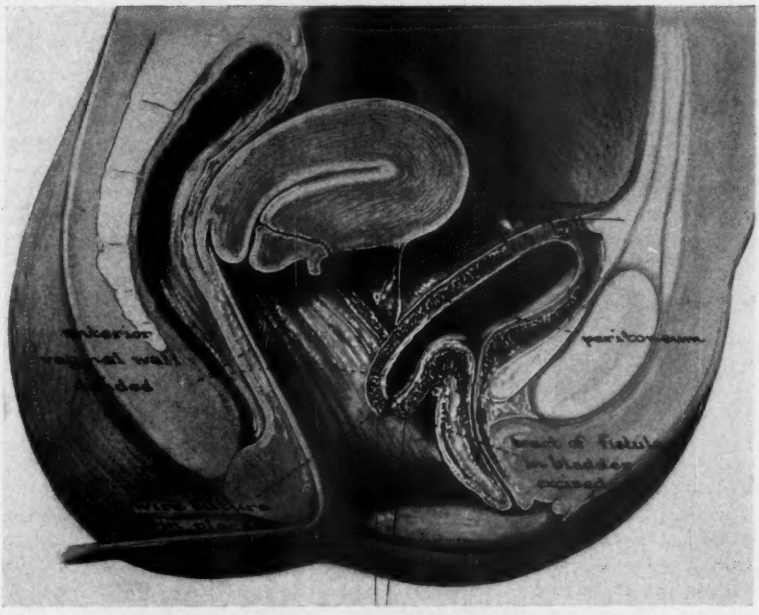

Fig. 3. Showing a method of layer closure for vesicovaginal fistulas with suture lines staggered. Often an additional intermediate row of interrupted alloy steel wire sutures are also introduced into the bladder.

With higher perforations through the rectovaginal septum, the margins of the opening should be excised, the rectal margins inverted and sutured from the vaginal side with fine alloy wire and a staggered layer closure of the vagina made, also with the wire. The anus should first be thoroughly divulsed to prevent back pressure. I prefer interrupted sutures, which interfere less with the blood supply than a purse string inversion of the rectal wall. For large obstructive fistulas with chronic vaginitis and proctitis, difficult to close otherwise, the lower rectovaginal partition is divided in the midline from the perineum to the fistula, scar tissue excised, strictures divided and sutured as in the Heineke-Mikulicz operation, and the edges of the rectum and vagina united with fine alloy steel wire, leaving the rectovaginal partition widely open.

After three to six months, during which time any tuberculous or syphilitic basis is carefully treated and all local reaction has subsided, the united rectovaginal margins are split open and carefully united in layers from the vaginal side with fine interrupted buried alloy steel sutures, taking care not to close the sphincters tightly. Occasionally, especially with a persistent local inflammation, preliminary colostomy is necessary. The following are illustrative cases:

In the first a successful closure with fine catgut sutures was obtained before the advantages of alloy steel wire were discovered.

Rectovaginal fistula following childbirth. Mrs. James K., aged 40, was admitted to Temple University Hospital, Jan. 3, 1932, having had a forceps delivery three months before, resulting in extensive perineal tears, for which immediate suture was done. The wound failed to unite and the patient had had loss of sphincter control and gas and fecal material had escaped through the vagina. A rectovaginal fistula about 1 cm. in diameter was found 2.5 cm. above the sphincters. At operation the perineum was split from the fistulous tract outward and the margins of the fistula excised. The vagina was closed by continuous submucous suture of continuous 000 catgut, the levatores ani united by interrupted 0 chromic sutures, and the divided rectal margins brought down and sutured to the skin with silkworm gut. There were no complications and the patient was discharged January 24, three weeks after operation.

Rectovaginal fistula. Miss Judith P., aged 26, admitted June, 1937, had developed a perirectal abscess in 1929, and had since been troubled with an offensive vaginal discharge. A rectovaginal fistula had been diagnosed in 1936, for which three unsuccessful operations had been performed. The patient had been troubled with frequent liquid stools without blood or mucus for 12 years, and more recently an afternoon elevation of temperature, hacking cough, and sputum occasionally showing a little blood. A roentgen diagnosis of bronchitis and ileocolitis had been made. There was a Y-shaped rectovaginal fistula extending on the rectal side from just above the internal sphincter to an opening on the posterior wall of the vagina about 3 cm. above the introitus.

On June 11 the fistulous tract was delineated by the injection of a solution of methylene blue, the perineum divided into the fistulous tract which was freely excised. The vagina and rectum were separated, sutured with buried interrupted sutures of No. 35 gauge alloy steel wire, the mobilized upper rectal margin being sutured to the margin of the skin. The levatores ani were united in the midline and the overlying triangular ligament and skin with layer interrupted sutures of alloy steel wire. To reduce pressure within the rectum the sphincters were not tightly closed. Primary union resulted. The following year the patient returned with a diagnosis of terminal ileitis, for which in a one stage operation about 100 cm. of the lower ileum was excised with end to end anastomosis. Primary intestinal union resulted and the patient was hospitalized only about two weeks.

Tuberculous rectovaginal fistula closed with wire after twelve unsuccessful operations. Miss C., 41 years old, with a history of tuberculous bone disease of the tibia for seven years in childhood, had had a high tuberculous rectovaginal fistula for twelve years. There had been twelve previous operations for the

fistula by various operators through the vagina, or abdomen, and including a left inguinal colostomy, and a hysterectomy. The latter was followed by widespread ulceration of the abdominal wall, lasting two years. In the Temple University Hospital the rectovaginal partition was first completely divided as described above with division of a tuberculous rectal stricture and the patient given intensive tuberculin treatment. After six months the rectovaginal partition was closed with buried layer sutures of fine alloy wire. Healing without reaction followed, and four weeks later the colostomy was closed. There has been no fecal leakage in the two years since the operation, although the patient thinks that an occasional bubble of gas enters the vagina without irritation.

It is especially in such a difficult case that the advantages of annealed alloy steel wire sutures are evident.

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CARCINOMA OF THE MALE BREAST

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IT is difficult to determine from a review of the literature what percentage of all breast cancers occur in the male because of the various methods of reporting cases. Some authors report a single case without any statistics as to the number of breasts examined. However J. T. Moore[1] in 1934 assembled a series of 56 cases in which statistics are given. In his own series of 739 breasts removed in Houston he found nine cases of carcinoma in men. In his total series, there were 56 cases in the male of a total of 7,468, or 0.75 per cent. A year earlier Neal[2] found 60 cases out of 9,279. If we are assuming that there is no overlapping in these two series, 116 cancers in the male were found in 16,747 cancers of the breast of both sexes. This is an approximate incidence of 0.7 per cent. Judd[3] reported 17 cases, Gilbert[4] 47, without giving further statistics as to the incidence.

In the opinion of these and other authors, from 0.7 to 2 per cent of all cancers of the breast occur in the male.

In my examination of 297 consecutive breast tumors, there were 115 benign tumors, 90 carcinomas and 2 sarcomas. Five of these tumors occurred in the male breast and four of them were benign. The fifth is reported here.

Report of Case

In January, 1935, a colored man, 66 years of age, was admitted to the City Hospital suffering with severe congestive heart failure. A small mass was noted in the lower outer quadrant of the left breast, seemingly well encapsulated, well attached to the skin but not attached to the ribs. It measured 2 by 2 by 1 cm. He stated that this mass had been present for approximately a year. It had been painless and slowly increasing in size. There was no history of trauma. The family history was irrelevant. Examination of the blood was without interest. The urine contained albumin and casts.

After the patient had improved under digitalis therapy, the mass, along with the skin and some of the underlying muscle, was excised under local anesthesia. On section of the tumor, increased resistance was encountered. The tumor was pearly white and grossly presented the typical appearance of cancer of the breast.

Microscopic Examination: The skin appeared normal but immediately beneath it there were large masses of epithelial cells which had the typical appearance of adenocarcinoma of the breast (fig. 1). Many of the gland acini were markedly dilated and filled with atypical embryonal epithelial cells, many of them showing mitotic figures. There was extensive infiltration into the stroma and in some areas a large amount of fibrous tissue production with strands of epithelial cells between the bundles. This area gave the typical picture of scirrhous carcinoma of the breast (fig. 2).

Fig. 1. Area of adenocarcinoma.

Fig. 2. Area of scirrhous carcinoma.

Follow-up: The patient returned to the out patient department about two months after operation at which time x-ray therapy was given to the anterior chest over the area of the scar. A total of 970 r units was given, using 125 KVP, 1 mm. Al, 0.25 mm. Cu filter, at 30 cm. distance. He was unwilling to return for further treatment.

He was readmitted to the hospital approximately nine months after the breast amputation on account of recurring heart failure. He died from this cause on October 16.

Autopsy: No evidence of recurrence could be demonstrated in the skin and no axillary glands or other metastases could be found. Upon opening the thorax the left lung was densely adherent in the apex and over the lateral

surface the pleura was thickened. In the upper medial aspect of the apex an air cyst approximately 5 cm. in diameter was present.

The heart was enlarged and the coronary arteries were tortuous and markedly sclerotic with a narrowing of the lumen due to atheromatous plaques. The kidneys were of the small red granular type.

SUMMARY

An additional case of carcinoma of the male breast is reported along with subsequent autopsy report six months after operation.

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BRONCHIAL TUMORS, DIAGNOSES AND TREATMENT

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THE subject of bronchogenic carcinoma is a broad one and the study of it is most interesting. Twenty-five years ago in only a small percent of the cases was the diagnosis made before autopsy. During the last ten or fifteen years progress has been made in both the diagnosis and treatment. There is occasionally considerable debate, even among experienced pathologists, as to whether a given tumor is benign or malignant. Some while histologically benign cause serious pulmonary complications which result in death as surely as if malignant. Therefore, it seems wise and timely to discuss both together. All bronchial tumors that have come under my observation in twelve months are included in this report. The period extends from Sept. 1, 1936, to Sept. 1, 1937. Numerous pitfalls encountered, theoretical origin of the tumors, pathology, histologic structure, and the classification have had to be omitted: the primary purpose of this discussion is to stimulate you to become tumor minded. The frequency of the disease merits its consideration as a possibility in all obscure pulmonary lesions. Its early diagnosis demands education of the public and close cooperation between the general practitioner, clinician, roentgenologist, laryngologist, and the surgeon.

INCIDENCE

Bronchogenic carcinoma until a few years ago was considered a rare disease but more recent study has shown it to be common. It ranks about third highest in frequency of all malignant tumors, varying from 6 to 11.4 per cent. Arkin[1] estimates 6 to 8 per cent. Rosedale and McKay[3] found 466 cancers out of 4,670 autopsies. There were 33 bronchogenic carcinomas which constituted 7.5 per cent of all cancers. This number, 33, was outnumbered only by cancers of the stomach, 71, and was equaled by cancers of the uterus. Jaffé[4] of Chicago in 1935 found 871 malignant growths in 6,800 autopsies. One hundred of these were bronchogenic carcinomas, constituting 11.4 per cent. This number ranked third highest in frequency (stomach 184, and intestines 118). In 1921 Jaffé and Sternberg[5] reported the data of 4,500 autopsies from Vienna, Austria. Bronchogenic carcinoma constituted 10.73 per cent of all malignant neoplasms found. Both of the latter reports were based upon large series. Both were made under similar conditions and by the same man. The

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two series were collected in different parts of the world, separated by a period of about fifteen years. They revealed percentages too constant to be considered accidental. This would indicate that the so-called increase in frequency is not real. It seems reasonable to assume that the apparent increase is due not to numerical increase in occurrence but rather to special study of the disease together with the alertness of the clinicians and the improved methods of diagnosis.

Benign bronchial tumors are more rare than malignant ones. Patterson[6] credits von Eicken[7] with the first bronchoscopic removal of a benign tumor. His report appeared in 1907. No other reports appeared in the literature so far as I know until Jackson's[8] in 1917.

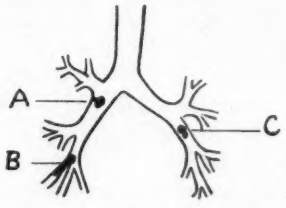

Fig. 1. Location of 3 benign tumors.

Since 1928 several articles by Reisner,[9] Wessler and Rabin,[10] Clerf,[11] Patterson,[6] Kramer[13] and others have directed attention to this condition. Jackson and Konzelmann's[12] recent report of 12 cases is instructive and stimulating. These data are convincing that benign neoplasms are also more common than previously believed. It is probable that some of the supposedly malignant tumors removed bronchoscopically were benign. Rabin and Moolten[15] offered an excellent criterion for differentiation of adenoma from malignancy.

Age. It is estimated that at least 75 per cent of all bronchogenic carcinomas occur between the ages of 40 and 60, with perhaps an equal distribution in the two decades. Eight of my sixteen patients were between 40 and 50 while six were between 50 and 60. Cancer is rare under 30 years of age; Edwards,[17] however, has reported two cases in a series of 73 patients.

Sex. There is a wide variation in the ratio of females to males; reports range from 1:11 + by Jaffé[4] to 1:3 + by Edwards.[17] In all series, males predominate. I have had 23 cases of bronchial tumors during one year. Four cases have been excluded from this analysis because the diagnosis was not confirmed. Though the excluded cases clinically, by x-ray and by bronchoscopic evidence were bronchogenic carcinomas, the necessary diagnostic study, to be discussed later, could not be done. This discussion is therefore limited to the remaining 19 patients whose diagnostic studies were completed and

a positive diagnosis reached in every instance. Sixteen of these were bronchogenic carcinomas and three were benign neoplasms. It is not implied that 15+ per cent of all bronchial tumors are benign. This series is too small from which to draw conclusions. If the four excluded cases were added to the 19 proven cases 13+ per cent would be obtained. This may not be greatly in error. See figure 1 for the location of the three benign tumors.

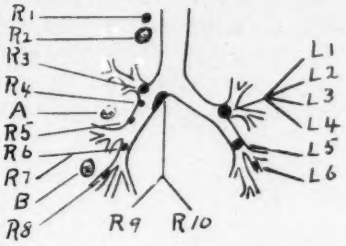

Fig. 2. Location of 16 bronchogenic carcinomas. R right, 10 cases. L left, 6 cases. A abscess in R7. B abscess in R8.

TABLE 1

3 Benign Bronchial Tumors

Case	*Sex*	*Age*	*Biopsy diagnosis*
*A	M	44	Inflammatory polyp
B	M	64	Benign papilloma
C	M	46	Inflammatory tissue

*Associated with mediastinal tumor—dermoid?

See figure 2 for the location of 16 bronchogenic carcinomas.

TABLE 2

16 Cases of Bronchogenic Carcinomas

Age	30-40	40-50	50-60	60-70
No.	1 (39)	8	6	1 (64)
Sex.....	All were males.			

It may seem strange that all patients in both series were males. This may be partly explained because about 60 per cent of the patients with tumor were in a hospital receiving predominantly males.

SYMPTOMS

The early symptoms of bronchial tumors, both benign and malignant, are due to bronchial irritation. The signs are due to bronchial stenosis and obstruction. In reality the signs are not of the tumor per se but of the complications—atelectasis, suppuration, abscess, effusion, etc. The earliest symptom is dry cough. The

cough may however, be productive of a scant amount of mucus. Christie[20] said, "there is nothing characteristic about it but when cough arises in a person at or beyond middle age and if persistent its serious possibilities should be kept in mind." It is unfortunate that many patients with tumor are treated several months, and often a year or more, as cases of bronchitis. Often the onset is with an acute severe respiratory infection and a diagnosis of pneumonia or influenza is made. Several weeks later, when convalescence is delayed, the roentgenogram is frequently interpreted as "unresolved pneumonia," "suppurative pneumonitis" or "abscess." If however, massive or lobar atelectasis with or without a superimposed infection, or if a tumor is seen, the correct diagnosis is likely to be made.

Fig. 3. Early bronchial type. Tumor does not occlude the bronchus.

Fig. 4. Late bronchial type. Upper lobe bronchus is occluded by tumor.

Hemoptysis occurs late in carcinoma, only after the bronchial mucosa has become ulcerated. However, according to Kramer,[13,14] hemoptysis is frequently the only and predominating symptom in benign tumors.

In my series of sixteen proven cases of bronchogenic carcinoma six had been diagnosed pulmonary tuberculosis (fig. 2: R4, R5, R6, R7, R9 and L1). Two patients (R6 and L1) had been in a tuberculosis sanitarium five and seven months respectively without positive sputum. R7 had been diagnosed tuberculous abscess without neoplasm being suspected. It is believed that from 10 to 15 per cent of pulmonary abscesses are due to bronchogenic carcinomas. In any case of non-tuberculous pulmonary suppurative lesion without a definite etiologic factor, the possibility of carcinoma should be considered. The signs and symptoms of bronchogenic carcinoma vary according to the location of the lesion and to the extent of bronchial, peribronchial and parenchymal involvement. They also vary according to the type, i. e., the bronchial or the peripheral. See figs. 3, 4, 5 and 6 for better interpretation of symptoms. Observe the variation in symptoms due to location and size of the lesion.

Signs and symptoms of the early bronchial type (fig. 3)

1. Cough.
2. Wheeze may or may not be present.

3. Routine roentgenogram is negative: however, inspiratory and expiratory x-rays may show a shift of the mediastinum. Fluoroscopy is of more value in detecting the mediastinal shift.
4. Bronchoscopy with biopsy is positive.
5. Lipiodol bronchogram may be of value if the bronchoscopy is not diagnostic.
6. Note the tumor does not completely occlude the bronchus.

Signs and symptoms of the late bronchial type (fig. 4)

1. Cough. Dyspnea may be present in varying degrees or may be absent.
2. Sputum is mucoid at first but later becomes foul or purulent.
3. Hemoptysis may be the cause of the patient seeking medical advice. This may occur before infection which produces signs of abscess.
4. Fever, pain, anemia, loss of weight, etc.
5. X-ray shows atelectasis, abscess, etc. Note elevated diaphragm in fig. 4.
6. Fluoroscopy shows shift of mediastinum with respiratory cycles. This mediastinal shift may be absent in the presence of mediastinal metastases.

Fig. 5. Early peripheral type.

Fig. 6. Late peripheral type. Note elevated diaphragm and deviated trachea.

Signs and symptoms of the early peripheral type (fig. 5)

1. Cough is the only symptom.
2. Weight loss is very little if any.
3. Pain may or may not be present.
4. Physical examination may be entirely negative.
5. Roentgenogram suggests pulmonary tuberculosis, abscess, pneumonitis, etc.
6. Diagnosis of unresolved pneumonia is frequent.

Signs and symptoms of the late peripheral type (fig. 6)

1. Cough is the predominating symptom. Dyspnea is usually present.
2. Sputum is foul, purulent or it may be streaked with blood.
3. Hemoptysis may or may not occur.
4. Pain, weakness, loss of weight and anemia.
5. Hoarseness, due to recurrent laryngeal nerve involvement, may occur.
6. Horner's syndrome may occur due to sympathetic nerve involvement.
7. X-ray shows pulmonary infiltration sometimes with atelectasis, elevated diaphragm, deviated trachea, abscess, etc.

Table 3 shows the symptomatic analysis of my sixteen cases of bronchogenic carcinomas, and also the diagnosis which had been made on these patients before admission to the hospital.

TABLE 3

Symptomatic Analysis of 16 Bronchogenic Carcinomas

Cough	16
Dyspnea (severe in 10, mild in 4)	14
Pain	13
(mild, admitted on questioning 4)	
(moderate, voluntary 3)	
(severe 6)	
Blood streaked sputum	7
Hemoptysis, included in the above	3
Onset as pneumonia or influenza	6
Loss of weight (complaint)	3
Wheeze	1
Diagnosis Before Admission	
Pulmonary tuberculosis	6
Bronchogenic carcinoma	3
Carcinoma suspected	2
Others	5
Carcinoma and suspected carcinoma	5
Carcinoma not suspected	11

Four of the six patients who had severe pain had abscess formation; three of the four patients with abscess had distant metastases. The two who did not have abscesses had metastases to both axillary and cervical lymph glands. This analysis indicates that severe pain signifies a far advanced lesion with probable metastasis or complication or both.

DIAGNOSTIC STUDY

Roentgenograms. X-rays of the chest are imperative but x-ray studies should not be limited to the chest. In a search for metastases x-rays should include the gastro-intestinal and genito-urinary tracts, long bones, pelvis, skull, spine, ribs, scapulae, etc., if there is any clinical evidence of involvement. It is not within the scope of this paper to go thoroughly into the x-ray characteristics of bronchial tumors. However, I have selected seven factors which should arouse suspicion of tumor with complications.

1. *Parenchymal infiltration.* There is a varying degree of surrounding pneumonitis and infiltration. The pus contains pyogenic organisms but not tumor cells. There may or may not be actual excavation. If a cavity is present it is more or less irregular in outline. Pathologically, cross section shows a smooth surface, red in color, and a clean cavity after the pus is evacuated. A fluid level is seen in probably 30 to 35 per cent of the cases.

2. *Infiltration.* This is manifested principally by radiating strands of increased density extending out from the hilum and is one of the first x-ray findings of tumor. If a malignant tumor is

present the strands are marked and can be seen to increase in density as the tumor grows. These are presumably due to (a) infiltration and actual growth of the tumor into the surrounding structure, (b) blockage of the peribronchial lymphatics, and (c) interference with circulation. In Volderaur's[18] series, this infiltration was present in 12 out of 16 squamous cell carcinomas, 12 out of 17 adenocarcinomas and in 7 out of 12 undifferentiated cell carcinomas.

3. Tumor. A mass is more often seen in the adenocarcinoma and perhaps in the undifferentiated cell carcinoma than in the squamous cell type. It was seen in three instances in this series, all of which were peripheral lesions (fig. 2: R1, R2 and L6).

Fig. 7. A, B and C show the bronchoscopic appearance on 5-19-37, 6-2-37 and 6-16-37 respectively. Note rapid growth of tumor and corresponding increase in size of abscess.

4. Atelectasis. This is a definite characteristic of the squamous cell carcinoma. In another series[18] it was present in 11 out of 16, while 2 others had no x-ray evidence of pulmonary lesion, as against 3 out of 17 adenocarcinomas and 4 out of 12 undifferentiated cell carcinomas. It was present in varying degrees in 10 of my 16 bronchogenic carcinomas.

5. Neoplastic abscess. The term neoplastic abscess designates central necrosis of the tumor. A superimposed infection in the center sooner or later takes place in the atelectatic area. This is in contrast to pyogenic abscess occurring in pulmonary tissue which is not invaded by neoplasm, but which has undergone suppuration. The neoplastic abscess is more round in outline. Pathologically it contains necrotic tissue, pyogenic organisms, and malignant cells. Cross section shows the thick walls to be rough and studded with small granular tumors and incomplete trabeculae that have not undergone necrosis.

6. Pleural effusion. This is present earlier in the peripheral than in the bronchial type. It is probably due to a pleuritis over a peripheral lesion or to blockage of lymphatics or to pleural or mediastinal metastasis.

7. *Metastases.* Local metastasis to the pleura and to the regional and mediastinal lymph glands is frequently obscured by pleural effusion and parenchymal infiltration.

If the pathologic behavior of these various cell types is considered the above roentgenologic findings are easly understood. The lowly malignant squamous cell carcinoma grows slowly and causes bronchial obstruction by proliferation and ulceration or stenosis due

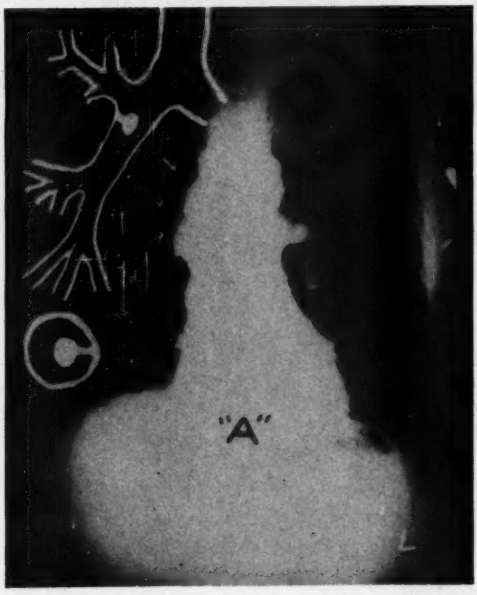

Fig. 8. Roentgenogram of Patient "A" showing mediastinal tumor, presumably a dermoid. Inset shows bronchoscopic findings of an inflammatory polyp of the right bronchus.

to submucosal infiltration and subsequent atelectasis. The adenocarcinoma grows rapidly with infiltration, is highly malignant and metastasizes early. The undifferentiated cell carcinoma has no characteristic features but may assume those of either the squamous cell or adenocarcinoma.

Bronchoscopy

Many clinicians readily refer patients with dysuria, frequency or hematuria to a urologist for cystoscopy but reluctantly refer patients to a thoracic surgeon for bronchoscopy. It is conservatively estimated that 75 per cent of all bronchogenic carcinomas can be seen bronchoscopically. Seventeen of the 19 patients with bronchial tumors were examined with a bronchoscope. Bronchoscopy with biopsy was definitely diagnostic in 14 cases or 82+ per cent. The

three patients (fig. 2: R2, R8, and L6) in whom a tumor was not seen had bronchoscopic findings sufficient to strengthen the clinical diagnosis. The diagnosis was confirmed by surgery in two cases, R2 and R8. Both had large abscesses. Excision of an axillary lymph gland confirmed the diagnosis in L6. When the tumor cannot be seen, bronchoscopic evidence suggestive of malignancy is fixation and rigidity of the bronchial tree, failure of the bronchi to shorten, elongate, contract or expand with the cycles of respiration, and widening of the carina. The two patients who did not have bronchoscopy (fig. 2: R1 and R5) were diagnosed respectively by aspiration biopsy, R1, and by excision of axillary lymph gland, R5. All three of the benign tumors were correctly diagnosed bronchoscopically.

DIFFERENTIAL BRONCHOSCOPIC APPEARANCE OF TUMORS

Malignant	*Benign*
Granular, rough, dull. May be ulcerated. May be covered with necrotic membrane. Usually vascular, may resemble granulating tissue.	Reddish, pink or yellow. Moist smooth, glistening surface. Sometimes rough.
Broad base, sessile, always embedded.	May be sessile, usually pedunculated, rarely if ever embedded.
Shape—usually flat unless completely fills bronchus.	Usually round, globular or oval.
Bronchial mucosa — always infiltrated.	Rarely if ever infiltrated.
Bronchus—later sign—fixation, rigidity, deformity, narrowed lumen. Does not contract, dilate, shorten and elongate with respiratory cycles.	Bronchus usually movable unless atelectasis with superimposed infection exists.
Carina—later sign—frequently widened, distorted, fixed, rigid. May not be in vertical position, displaced.	Normal unless involved by the tumor. Shift toward or from diseased side with respiratory cycles depending on partial inspiratory or expiratory block.

Other bronchial lesions are sufficiently obvious that little difficulty is experienced in differentiating them from tumors.

Considering the above differential points and the diagnostic value of bronchoscopy it is obvious that the bronchoscope is indispensable. I advise a carefully performed diagnostic bronchoscopy on every patient with unexplained cough, hemoptysis or wheeze and every case of non-tuberculous suppurative pulmonary lesion. The idea—

"wait and see what happens"—cannot be too strongly condemned. Clerf[21] said, "Next to its usefulness in foreign bodies in the air passages bronchoscopy has contributed more to our knowledge of pulmonary carcinoma than any other procedure." It must be borne in mind that a cough is not always bronchitis, an hemoptysis is not always tuberculosis and a wheeze is not always asthma. Although Father Time is considered the Great Healer in the majority of diseases, he becomes the Great Destroyer in bronchogenic carcinomas. One patient (fig. 2: R7), who was obviously inoperable was followed bronchoscopically from his admission to the hospital four months after the onset of cough and two months after hemoptysis. The bronchoscopic findings are shown in fig. 7. Observe the rapidity of tumor growth and the increase in size of the abscess. Death from metastases to the brain, and lung abscess occurred about one month after the last bronchoscopy.

It is not implied that bronchoscopy nor bronchoscopy with biopsy is conclusive. The final verdict comes from the pathologist. Occasionally numerous sections are required before a verdict may be reached. The pathologist's report from the first biopsy is frequently "inflammatory—no evidence of tumor." This neither confirms nor denies the presence of a tumor. It demands another bronchoscopy which should be done within a week or ten days. Four of my cases necessitated more than one bronchoscopy before a definite diagnosis was established. The tumor may not be seen at the first examination but as growth proceeds it protrudes through a small bronchus and is readily visible. Bronchoscopy, showing as it does the location of the tumor as well as its extent, is a valuable aid to the surgeon in deciding whether or not the condition is operable.

Aspiration biopsy. If a tumor is known to be against the thoracic wall and if the x-rays show a very thick pleura associated with it, an aspiration biopsy may safely be done. This was used once in this series and was diagnostic of a bronchogenic adenocarcinoma. The method must not be utilized in the presence of infection if good pulmonary tissue has to be penetrated in reaching the tumor.

Bronchogram. If bronchoscopy with biopsy is diagnostic, a bronchogram with lipiodol is not necessary. Otherwise the bronchogram with posterior-anterior, lateral, right and left anterior oblique x-ray projections often render valuable information. In such instances I permit the lipiodol to go only into the diseased side, except a small amount into the other stem bronchus so that widening of the carina can be detected.

Surgical biopsy. When an enlarged lymph gland is found associated with either a known carcinoma or obscure pulmonary lesion the gland should be removed for histologic study. This procedure

was diagnostic in two of these cases; in one no bronchoscopy was done and in the other careful bronchoscopic search failed to reveal tumor.

Pleural effusion. Fluid frequently masks interpretation of parenchymal involvement in the roentgenogram. When present it should

Fig. 9. Microphotograph of the inflammatory bronchial polyp from Patient "A," figure 8. Observe the papillary arrangement of the mucosa. This should not histologically be classed as a true neoplasm. It indicates tissue reaction to inflammation.

be aspirated and examined for cancer cells. The fluid may be clear or straw-colored when recent but becomes bloody or blood-tinged later in the disease. Replacement of fluid with air and additional x-rays frequently aids in detection of pleural nodules and metastases.

Sputum. If the sputum contains necrotic tissue or is thick and purulent, provided the above methods are not diagnostic, it should be examined for cancer cells. Sputum analysis has not been of any diagnostic aid in my experience. Theoretically it should be of value in advanced cases. It is used frequently in England with some success. Dudgeon and Wrigley[16] report sputum examination positive

in 68 per cent of their proven bronchogenic carcinomas. This, however, would not be present in early cases.

Pneumothorax. Introduction of air into the pleura permits better visualization of the mediastinum and pleura and thereby aids in the detection of metastases. It shows pleuropulmonary adhesions, when

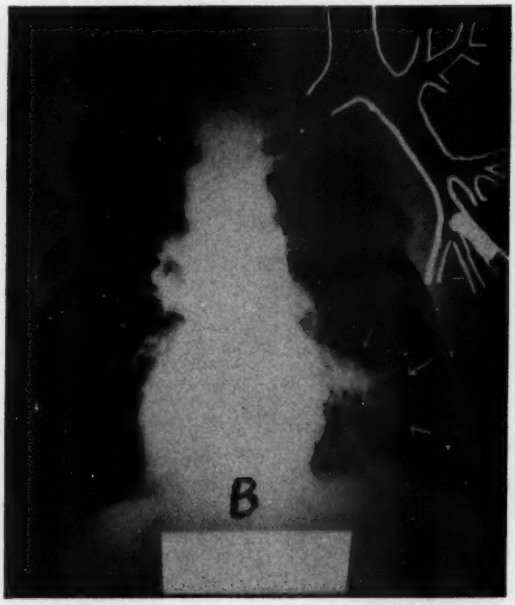

Fig. 10. Roentgenogram of Patient "B" showing atelectasis in right base. Inset shows bronchoscopic findings of a benign papilloma.

present, and aids the surgeon in evaluating the patient as an operative risk. It gives an idea as to whether operation may be simple, difficult or virtually impossible. Together with other tests it aids the surgeon to estimate the respiratory reserve of the patient. When pneumothorax is not well tolerated clinically, because of dyspnea, it is reasonable to assume that additional burden incident to surgical removal of the entire lung would probably be fatal in a one-stage operation. Some of these may possibly be successful if done in two stages.

If the diagnostic pneumothorax is well tolerated it should be continued for a week or ten days before performing pneumonectomy. During this time certain beneficial effects are accomplished; (1) it tends to prevent circulatory and respiratory disturbance upon opening the pleura, and (2) it greatly facilitates the operation and lessens shock.

Thoracoscopy. This may be of value in peripheral lesions provided adhesions do not prevent preliminary pneumothorax. In the absence of infection of the parenchyma section for biopsy may be taken. Possible development of empyema following biopsy from a tumor within infected tissue must not be ignored.

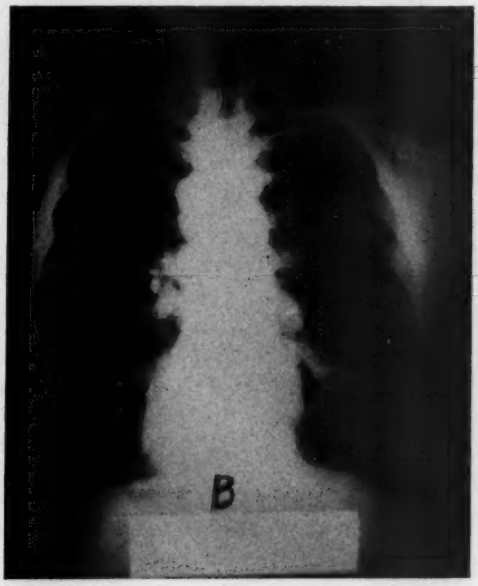

Fig. 11. Same patient as Figure 10. Note clearing of atelectasis in right base six months after bronchoscopic removal of all accessible papillomas.

Search for metastases. Aside from the x-rays mentioned above careful physical examination for enlarged lymph glands should be made. Careful neurologic examination is done to rule out brain metastases.

Exploratory thoracotomy. This is used only if the diagnosis cannot otherwise be made. If skillfully performed it is not associated with any greater danger than an exploratory laparotomy. Proper location of the exploratory incision permits dealing with the situation according to indications. I believe exploratory thoracotomy is not done as often as it should be. Intrathoracic tumors should be explored when a diagnosis cannot otherwise be made, provided definite contraindications do not exist.

Table 4 shows a diagnostic summary of my 19 bronchial tumors.

TABLE 4

Diagnostic Summary of 19 Bronchial Tumors

Bronchoscopy with biopsy	14
Aspiration biopsy (no bronchoscopy)	1
Lymph gland (1 with, 1 without bronchoscopy)	2
Surgical (both abscesses)	2
Total	19
Patients bronchoscoped	17
Patients not bronchoscoped	2
Total	19
Bronchoscopic diagnosis	82+%

OPERABILITY

Is the case operable? Sometimes this question can be answered only after the chest has been opened. Unfortunately there is no method whereby the ability of a patient to tolerate a major surgical procedure can be accurately measured. Distant metastases obviously preclude surgery. Involvement of the carina rendering closure of the bronchial stump impossible contraindicates pneumonectomy. Further study is necessary for complete solution of this important question. In any case surgical judgment of the operator is a great factor. This judgment is good or bad in direct proportion to the surgeon's ability accurately to interpret the diagnostic studies and just as accurately to evaluate his own skill and experience in handling the difficulties indicated by these studies.

PREOPERATIVE PREPARATION

The importance of adequate preoperative preparation cannot be too strongly emphasized. The operation itself is a hazardous procedure and the disease for which it is done is a grave one. Usually the patient has already been weakened by complications such as atelectasis and superimposed infection. Careful attention should be directed to the fluid balance. The routine urine and blood studies should be done. Transfusion of blood is done routinely, several times if necessary. Electrocardiographic studies should be made. The morning of and just before operation a cannula should be inserted into a vein, preferably at the ankle, so that intravenous fluid, blood or medication may be given as indicated.

TREATMENT

Benign neoplasms. These can usually, though not always,[28] be removed in one or more sittings bronchoscopically. If peroral removal cannot be done lobectomy or pneumonectomy should be con-

sidered depending upon the location, and the severity of the symptoms. The three benign tumors in the present series (fig. 1, table 1) were treated bronchoscopically. Patients A and B had four bronchoscopies each. In patient A the bronchial tumor was found incidental to a diagnostic bronchoscopy for a dry persistent cough

Fig. 12. Microphotograph of benign papilloma from Patient "B" shown in Figures 10 and 11. Compare this true neoplasm with Figure 9 showing inflammatory polyp.

associated with a mediastinal tumor, presumably a dermoid. The inflammatory polyp was removed by the piecemeal method seven and a half months ago and the patient is still free of symptoms. The mediastinal tumor may be removed later. Patient B ten months after piecemeal bronchoscopic removal of all accessible papillomas is greatly improved. He has gained weight and feels well but has a slight residual cough. The x-ray shows marked clearing of the atelectasis. It is however improbable that all the tumor was removed because of its location far down in the smaller bronchi. If this patient were much younger (he is 64) lobectomy would be advisable. Placing a radium needle within the lumen of the bronchus, but not into the pulmonary tissue, may be attempted later if his con-

dition should become worse. The tumor in patient C (fig. 1, table 1), which histologically showed only inflammatory tissue, was removed at one sitting eight and a half months ago. A bronchoscopy three weeks later showed no evidence of remaining tumor. He has not returned for check-up examination. Benign tumors frequently

Fig. 13. Roentgenogram of Patient "L2." Note complete atelectasis of left upper lobe with deviated trachea toward the left. Inset shows bronchogenic carcinoma occluding the left upper lobe bronchus.

become malignant later. Because of pulmonary complications resulting in death, benign tumors should be removed early.

Malignant Neoplasm. The poor results with roentgen-ray and radium therapy in past years has greatly stimulated the surgical management of bronchogenic carcinoma. So far as I know the only proven case cured by x-ray therapy was reported by Christie.[19] According to him the case will be reported in detail later by Col. Wm. L. Keller. At present the practical problem is early diagnosis, and the treatment is surgical.

Surgical treatment may be palliative or radical. One of my patients (fig. 2 : R1), obviously had an inoperable bronchogenic adenocarcinoma with the classical symptoms of a Pancoast[26] or superior pulmonary sulcus tumor. Pain was intense. A cervical cordotomy was done. He lived six months in relative comfort.

The technic for lobectomy or for pneumonectomy is not standardized yet. Within the last few years a number of writers, Alexander,[27] Graham,[28] Haight,[29,30] Overholt,[31,33] Churchill,[34,35] Reinhoff,[24,25] Longacre[36] and others have made contributions to the technic of the operation. Both operations are now accepted pro-

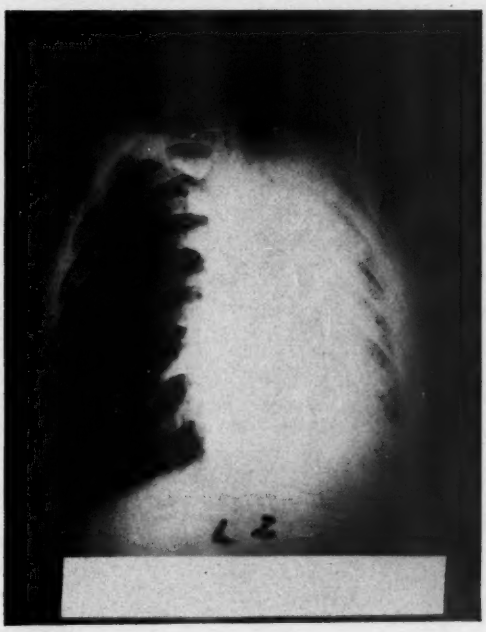

Fig. 14. Roentgenogram of Patient "L2" made 26 days after removal of entire left lung. The restitutional mechanism has almost obliterated the left hemithorax.

cedures throughout the world. Each patient, as well as the underlying pathologic findings, presents an individual problem and the technic must vary according to the indications. One is not justified in deciding before the operation whether or not a one-stage or a two-stage operation will be done. These problems can be solved only after the chest has been opened. Lobectomy versus pneumonectomy for bronchogenic carcinoma, in certain locations, is a subject of considerable debate among thoracic surgeons. I do not believe lobectomy should ever be done for a carcinoma that can be seen with the bronchoscope. Because of the anatomic relationship of the lymphatics, blood vessels and structure of the lung and tracheobronchial tree, I believe lobectomy bears a similar relationship to pneumonectomy that simple excision of breast carcinoma bears to radical mastectomy.

Sixteen of the 19 cases in this series were proven bronchogenic carcinomas. Only 3 of the 16 patients were considered to be fair surgical risks and one of these was questionable. The remaining 13 patients were obviously inoperable because of metastases, age, general debility, etc. One patient (fig. 2: R6) had a lung which was

Fig. 15. Patient "L2" three months after removal of entire left lung. Note the relative absence of chest wall deformity.

almost entirely adherent to the parietal pleura, especially posteriorly and around the base. It was technically very difficult to separate. He died forty-eight hours after the operation. The other two patients (fig. 2: L1 and L2), were surgically successful. Owing to adhesions both operations were difficult. L1 had a neoplastic abscess in the periphery of the lung next to the mediastinum. It was inadvertently ruptured, infecting the pleural cavity. Pneumonectomy was done and dependent drainage established. His postoperative reaction was excellent for 12 days. A pyo-pericardium due to streptococcus mucosa (pneumococcus type 3) developed. The pericardium was successfully drained and again he did well. Nine days later, 21 days after the pneumonectomy, he died suddenly of a hemorrhage due to necrosis of the severed end of the pulmonary artery. The other case, L2, had a total pneumonectomy eight and a

half months ago. He is doing well with no evidence of metastasis. His appetite is good and he has gained weight. His cough and sputum have completely disappeared. Because his restitutional changes have been somewhat slow he is slightly dyspneic on exertion. However, his general condition is considered excellent.

The operative approach for pneumonectomy varies with different operators. A high anterior approach was used in this series with satisfactory exposure. No rib was removed. Complete restoration of the thoracic cage was accomplished. Because of obvious infection the pleural cavity was drained in all cases by the water-seal method.

Postoperative Treatment

Careful postoperative management of these cases is of paramount importance. Intranasal oxygen or an oxygen tent is used in every instance. I prefer the former because it facilitates nursing care. The fluid balance must be maintained. Although shock is minimal, or totally absent, except in the difficult cases, blood transfusions are given routinely, repeatedly if necessary. Intravenous fluid should always be given slowly. The heart should not be overloaded with rapid intravenous injections of large quantities of fluid. I believe that this was a contributing, if not the sole, cause of the loss of one patient. As only one lung is left, the air passages must be kept clear by encouraging the patient to cough up secretions which are prone to collect in the tracheobronchial tree. Finally, the surgeon must fully appreciate the importance of infinite care during the postoperative period. Slipshod methods and poor judgment in the days following pneumonectomy or lobectomy undoubtedly cost lives which good surgery should otherwise have salvaged.

Results

Results in the three benign tumors in this series were entirely satisfactory.

The three pneumonectomies for bronchogenic carcinoma of the one year series reported show an immediate surgical mortality of 33.3 per cent and a late mortality of 66.6 per cent. These figures are misleading. The cases were advanced and consequently bad surgical risks. The mortality rate based upon several years experience and including different writers is about 40 per cent. Early diagnosis should greatly improve these figures. If carcinoma of the lung is to be dealt with successfully treatment should be instituted before appearance of late symptoms such as pain, dyspnea, hemoptysis and loss of weight. Benign tumors must be sought for and removed early to prevent fatal complications.

Conclusions

1. Nineteen cases of bronchial neoplasms are reported. Sixteen of these were malignant and three were benign.

2. A symptomatic analysis is made.

3. A diagnostic study is outlined.

4. The early need for, and the value of bronchoscopy are emphasized. Correct diagnosis was made in more than 82 per cent of the patients upon whom bronchoscopy was done. Bronchoscopic evidence without seeing the actual tumor is valuable.

5. Roentgenogram findings that should arouse suspicion of bronchial tumor are mentioned.

6. Lobectomy versus pneumonectomy is discussed.

7. The importance of preoperative and postoperative management is emphasized.

8. Necessity for cooperation between the general practitioner and the specialist in making early diagnosis is discussed.

9. Three cases of total pneumonectomy for bronchogenic carcinoma and three cases of benign bronchial tumors removed perorally are reported.

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THE SURGICAL MANAGEMENT OF INCOMPETENT GENITAL SUPPORTS IN THE FEMALE

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Atlanta

A YEAR ago an effort was made to demonstrate the supports mainly of the uterus in a diagrammatic way, to explain the mechanics of these supports and a plea was made to apply the principles of mechanics in the treatment of altered function due to stretching or tearing of these supports.[1]

In a second paper[2] the perineal supports were illustrated diagrammatically and the importance of the perineal body and the urogenital trigone as the main supports of the perineum was emphasized. Again a plea was made to observe physiologic, anatomic and mechanical principles in the surgical correction of altered mechanics of these supports.

This paper is a continuation of the other two, giving the surgical management of injured supports based upon anatomic, physiologic and mechanical principles.

The main supports of the uterus are the uterosacral and uteropubic, (fig. 1a) and the cardinal ligaments (fig. 1b) arranged in a crossbeam fashion, with the cervical portion of the uterus occupying the intersection of these supports in the center of the pelvic cavity in the superior plane level (fig. 2). The uterus is so placed in this crossbeam arrangement that the fundus normally is directed upward and forward and the cervix downward and backward with its long axis at right angles to the vaginal canal. In this position intra-abdominal pressure is directed against the posterior surface of the fundus, which aids in holding the fundus flexed forward. As long as the integrity of the supports is maintained the uterus will remain in this position. If, however, these supports are damaged and stretched, the cervix will occupy a lower position in the vaginal space, the fundus will rotate backward and downward, and thus the intra-abdominal pressure is changed from the back to the front of the fundus. This pressure continued over a period of time forces the uterus down toward the inferior plane so that retroversion and prolapse result. The degree of stretching of ligaments determines the degree of prolapsus and this depends largely upon the amount of intra-abdominal pressure. The occupation, habits and temperament of the individual are factors to be considered in the production of intra-abdominal pressure. The physical condition of the individual must be considered in evaluating the integrity of supports

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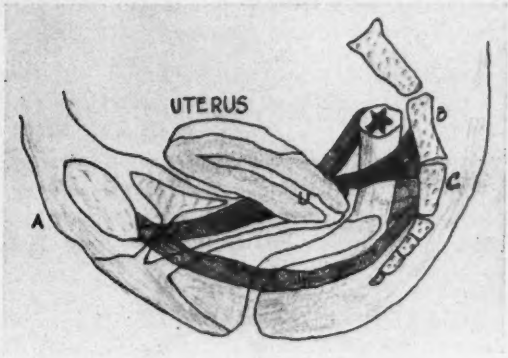

Fig. 1 (a). AB indicates the sagittal supports (the uterosacral and uteropubic segments) of the uterus in the superior plane. AC indicates the sagittal support in the inferior plane (the levator ani muscles and their fascia). Modified from Crossen's textbook.

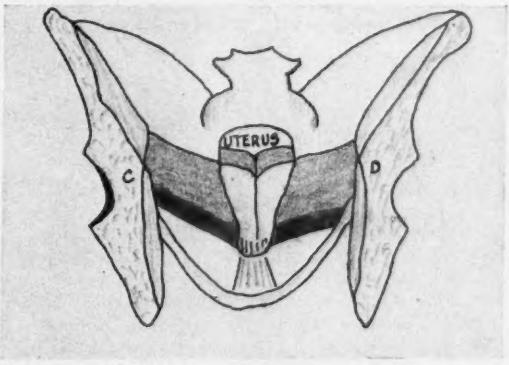

Fig. 1 (b). The transverse supports (the cardinal ligaments, the shaded portion at the base of the broad ligament) C. D. Modified from Crossen's textbook.

to withstand pressure. Congenital defects must also be considered. A woman who does constantly hard physical labor has a greater amount of intra-abdominal pressure. One who suffers from constipation is constantly subjected to increased intra-abdominal pressure. A frail, poorly nourished woman or one who suffers from constitutional disease is more likely to have weakened supports. Supports may be poorly developed, that is, they may be congenitally weak. All of these factors should be evaluated in planning the management or treatment of altered or faulty mechanics of supporting structures.

It may be necessary to remove a diseased uterus or it may be best to interpose the uterus. One of the ventral fixation operations such as the bisection suspension, or the Hertzler type suspension, etc., may be decided on because of certain indications. A Leforte may

be selected for good reasons. Each case must be individualized and the type operation chosen to suit the case.

The operative treatment for retroversion is the treatment for prolapsus, except in those cases of retroversion due to pelvic inflammatory disease and not due to damaged supports. Moderate prolapsus may be corrected by shortening either the uterosacral or the cardinal ligaments. In marked prolapsus, however, both the uterosacral and cardinal ligaments should be shortened (figs. 3a and 3b). The round ligaments may be shortened only for the purpose of

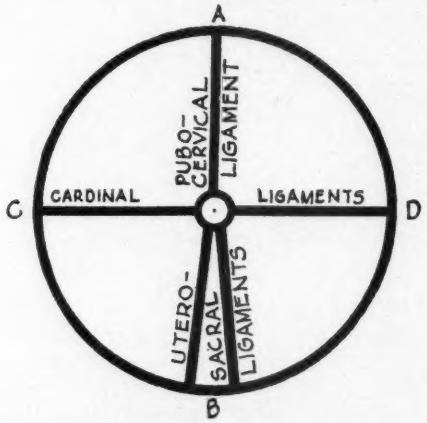

Fig. 2. The crossbeam arrangement of the supports of the uterus in the superior plane. AB, the sagittal supports (the uterosacral ligaments and uteropubic ligament). CD, the transverse supports (the cardinal ligaments) (Diagrammatic).

aiding in holding the fundus forward. Emphasis should be placed upon the cause of the prolapsus and therefore correct the causative factor as nearly as possible. George Gray Ward stated in his address before the British Congress of Obstetrics and Gynecology in Belfast, Ireland, April 2, 1936, "The principle of reefing the over stretched cardinal ligaments of the upper pelvic floor, by approximating them in front of the cervix or its stump, if amputated, thus elevating and anteverting the uterus by throwing backward the lower pole of the organ is a sine qua non for the cure of its prolapse if that organ is to be conserved." He states further, "If marked prolapsus uteri and associated retroflexion are present, shortening of the utero-sacral and round ligaments is necessary." For the methods of shortening the uterosacral and the cardinal ligaments and the other technic employed in the Watkins interposition, the Leforte, and the different ventral fixations, reference is made to any good textbook on gynecology of recent publication and particularly to Dr. Ward's paper.

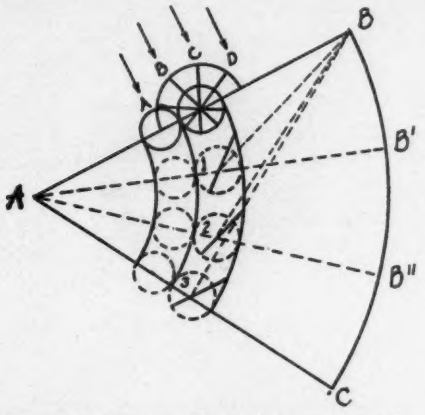

Fig. 3 (a). Diagrammatic representation of the normal position of the uterus in the superior plane level (A) and of the position of the uterus in the different degrees of descensus, 1, 2 and 3 in the broken circles, in the vaginal space, and the stretched uterosacral ligaments, 1, 2, 3, B broken lines. It is obvious that if the uterus is to be restored to its normal position in the superior plane and maintained there the stretched uterosacral ligaments B 1, 2, 3, must be shortened; the degree of shortening depending upon the degree of prolapsus.

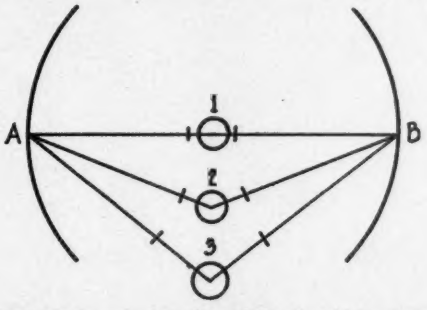

Fig. 3 (b). Diagrammatic representation of the stretching of the cardinal ligaments in descensus uteri, A-B, 1, 2, and 3. The stretched cardinal ligaments also must be shortened if the uterus is to be maintained in the superior plane level in prolapsus of second and third degree.

An uncomplicated retroversion of the uterus without symptoms is not a pathologic entity and requires no treatment. A congenital retroversion in the absence of pelvic pathology is normal and requires no gynecologic treatment. If symptoms are present such as backache, the treatment is orthopedic. Retroversion with adhesions or adnexal disease with adequate supports does not come within the scope of this paper. Retroversion with moderate prolapsus without symptoms may be treated satisfactorily with mechanical supports and postural exercise, or may need no treatment at all.

The supports of the perineal region are the urogenital trigone composed of fascial plates arranged one above the other (fig. 4),

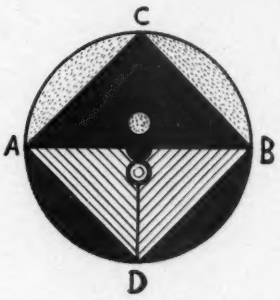

Fig. 4. PERINEAL SUPPORTS OR DIAPHRAGM. A-B-C, the urogenital trigone (the supporting fascia). A-B-D, the anal region. The levator ani muscle fibers are represented by the lines emerging from beneath the urogenital trigone and converging in the midline and to the coccyx. The perineal body is represented by the solid black figure in the center.

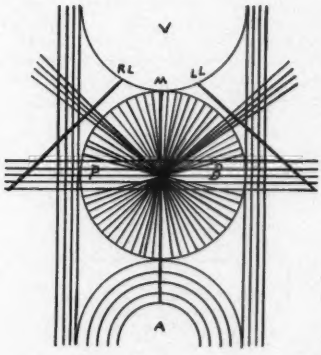

Fig. 5. PERINEAL BODY (ENLARGED). The radiating lines in the perineal body indicate diagrammatically the converging and interlacing fibers of the levator ani and transverse perinei muscles. The parallel lines on each side represent the fibers of the pubo-coccygeus muscles as they pass back beside the vagina, the perineal body and the rectum. The heavy lines, RL, MA, and LL indicate the sites of tears during delivery.

and a large central tendon the perineal body composed of the inserting fibers of the transverse perinei and levator ani muscles (fig. 5), placed between the vagina and rectum. The muscles of the perineal region have very little supporting power. Their function is to maintain perineal tone and to produce action which aids in defecation, voiding and parturition.

During the past quarter century obstetric management has been improved so greatly that very few perineal supports are left damaged. The practical abandonment of the use of powerful oxytocics and the obstetric forceps and the employment of the episiotomy incision have greatly minimized the incidence of damaged perineal supports. The same may be said of the supports of the uterus.

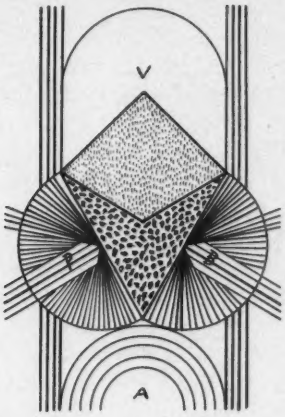

Fig. 6 (a). MEDIAN FIRST DEGREE TEAR (through the vaginal side of the perineal body and posterior vaginal wall). The dotted and mottled areas indicate the outline and extent of the tear at the time of tearing.

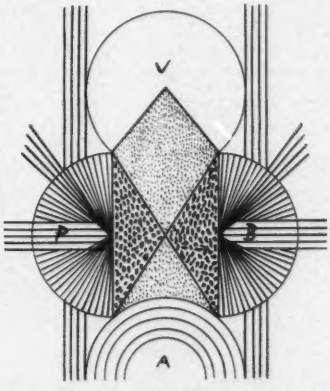

Fig. 6 (b). MEDIAN SECOND DEGREE TEAR (through the perineal body down to the sphincter ani muscle). The torn segments of the perineal body are pulled apart as indicated by the mottled area. The extent and outline of the tear is indicated by the dotted and mottled areas.

Proper after delivery care and the employment of postural exercise and properly fitted mechanical supports to a sagging uterus have greatly reduced the incidence of uterine displacements. No longer do you see in the clinics of the best surgeons the long list of cases posted for perineorrhaphies and suspensions. Only in cases of non-union or neglect in repairing tears does it become necessary to do secondary repairs of the perineum.

Tears usually occur in the midline in or through the perineal body and are graded first, second and third degree tears. A tear that extends into the perineal body but not completely through it is a first degree tear. One that extends completely through the perineal

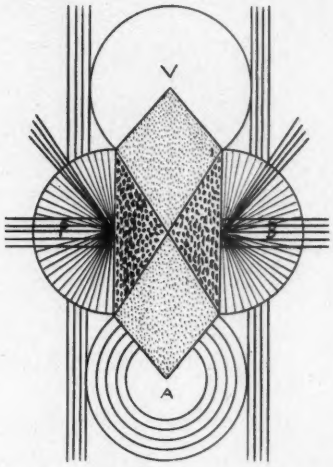

Fig. 6 (c). MEDIAN THIRD DEGREE TEAR (through the perineal body and anterior rectal wall and sphincter ani muscle fibers). The two triangular areas represent the torn perineal body segments which are drawn apart. The two irregular diamond-shaped areas indicate the outline of the tears in the posterior vaginal and anterior rectal walls and perineal space.

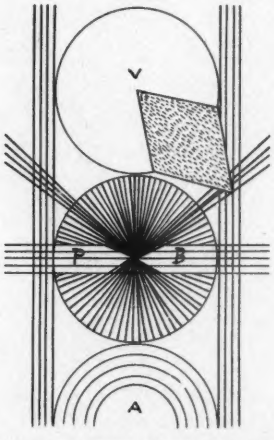

Fig. 7. LATERAL TEAR IN ITS RELATION TO THE PERINEAL BODY. The diamond-shaped area indicates the extent and outline of a tear at the time of tearing.

body to the sphincter ani muscle is a second degree tear and one that extends through the sphincter muscle into the rectum is a third degree tear.

In a median tear of the first degree, the posterior vaginal wall, the skin, and the vaginal side of the perineal body are damaged. The segments of the perineal body are only partially displaced to

the sides (fig. 6a). In second and third degree tears the torn segments of the perineal body are displaced and pulled away from the midline due to contraction of the fibers of the transverse perinei and levator ani muscles (fig. 6b). In third degree tears the sphincter ani muscle and anterior rectal wall are torn. The ends of the torn sphincter muscle retract and pull with them the torn edges of the rectal wall (fig. 6c). A tear may be lateral to the perineal body (fig. 7). The structures damaged in a lateral tear, depending upon the extent of the damage, are the vaginal wall and skin and the deeper structures such as the urogenital fascia, the pubococcygeus and transverse perinei muscles. Perineal body and fascia tears may occur although there is no tear in the skin or vaginal wall with the familiar deformities such as rectocele and cystocele and gaping introitus resulting.

In the correction of these defects all damaged structures should be restored, that is, the torn vaginal and rectal walls, the urogenital fascia, the torn sphincter ani muscle and the perineal body segments and skin should be properly approximated and sutured, whether the repair is done immediately or later. In the interval operation, scar tissue must be denuded. The outline of the old tear can be seen on close inspection after the parts have been painted with mercurochrome, iodine or tincture of metaphen. After scar tissue is denuded the wound is to all intents and purposes converted into the original tear and should be closed as such. Rectovaginal and vesicovaginal fascia tears should be repaired for the correction of rectocele and cystocele and the perineal body repaired on the same principle as in an open tear of that structure.

Before an operation is planned the integrity of the uterine, bladder, perineal and rectal supports should be properly evaluated. The pubococcygeal, transverse perinei and sphincter muscles should be tested, the cervix inspected and the uterus and adnexa palpated. The lumbosacral index should be measured to determine the superior plane incline. Only after these observations have been made can an operative procedure be intelligently planned.

The technic in a first degree tear is carried out as follows: The position of the patient and drapes are the same as for any vaginal operation. A very careful inspection of the damaged area is made for the outline of the old tear. When this is determined the upper angles of the tear on each side of the vagina and the angle in the midline in the vagina are picked up with Allis clamps and the tissue between these points is put on the stretch. The old tear is outlined with the knife and the tissue denuded, thus leaving a raw area which corresponds to the original tear. (Hegar's triangular method of

denudation.) If a fourth Allis clamp picks up the edge of the skin in the midline and traction is made in opposite directions an irregular diamond shaped area of denuded tissue is seen (fig. 8. Compare with fig. 6a). The segments of the perineal body, the urogenital fascia, the vaginal wall and skin are ready to be approximated. The

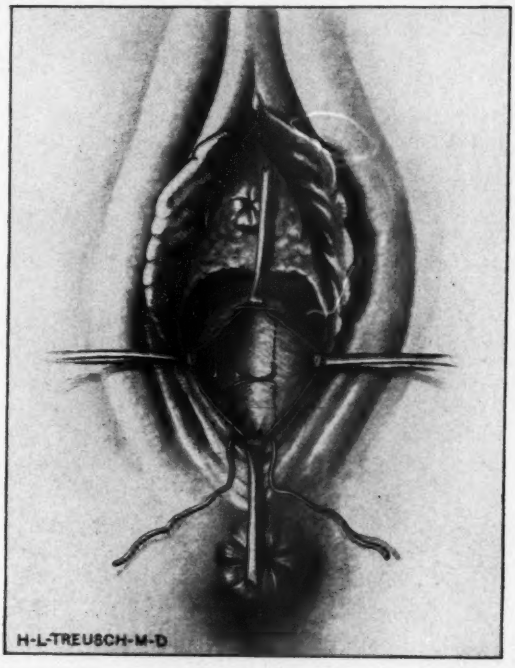

Fig. 8. Indicates the denuded area in a first degree tear and the first figure of 8 suture in place. Three other sutures are similarly placed for complete closure.

first stitch starts near the skin angle on the left, 0.5 cm. from the midline. It picks up the skin, fascia, and the rectal side of the perineal body and emerges the same distance from the midline halfway between the median skin angle and the median vaginal angle. It is then carried across to the opposite side and introduced at about the same level from which it emerged. The vaginal side of the perineal body and vaginal wall are included in this stitch. The needle emerges on the vaginal edge 0.5 cm. from the midline on the right side. It is then carried over to the left side and the vaginal edge and the vaginal side of the perineal body is picked up. The midline is crossed again and the rectal side of the perineal body, fascia and skin are included in the stitch, which completes the introduction of the first suture. It is noticed that the course taken by this suture is that of a figure of 8 (fig. 8). Other sutures are simi-

larly placed and tied. Very little tension is necessary to approximate completely the parts in tying the sutures. If a careful denudation is done and the sutures placed as described there will be no dead space left after the sutures are tightened and tied, thus minimizing hemorrhage, blood clots, swelling and pain. There is practically no con-

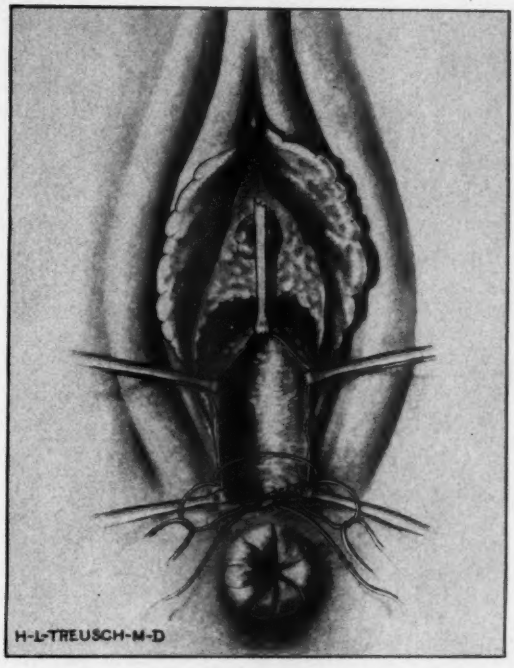

Fig. 9. Indicates the denuded area in a second degree tear and the figure of 8 suture placed to approximate the rectal side of the perineal body. When this suture is tightened, the wound is ready to be closed in the same manner as a first degree tear. (This is demonstrated more clearly in fig. 11 (a-b).

striction of tissue and the dangers of infection and sloughing, and breaking or cutting out of sutures are practically eliminated.

In second degree tears there is more tissue damage. The perineal body is completely divided and more damage done to the urogenital fascia. The rectovaginal fascia is usually torn and rectocele present. Very often the perineal body segments are drawn around to the sides of the sphincter muscle. In addition to the three clamps placed as in a first degree tear, two other clamps are placed, one on each side at the rectal margin. Traction is made in the opposite direction and the old scar outlined and denuded. The outline of the tear is found to be in the shape of a square with a triangle erected on the vaginal side (fig. 9: Compare with fig. 6b). The rectocele is re-

paired and redundant vaginal wall trimmed away and sutured. A suture is now placed in the skin margin and fascia of the sphincter muscle 0.5 cm. from the midline on the left. This suture crosses the midline and picks up the rectal edge of the perineal body, fascia, and skin, near the lower angle on the right. It is then carried back

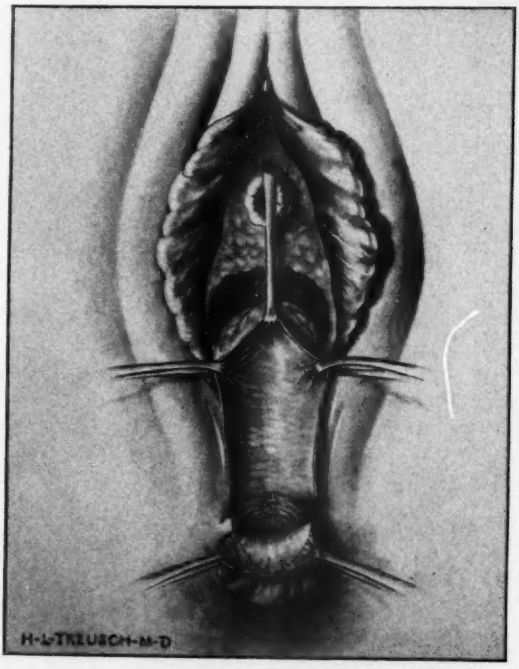

Fig. 10. Indicates the denudation of the vaginal portion of the scar tissue and the flap turned down over the rectal portion in a third degree tear; also the sutures placed in the sphincter ani muscle. The dotted line in the flap shows where the flap is trimmed off ready for closure.

across to the opposite side and introduced in the reverse manner, picking up the skin, fascia and edge of the perineal body and lastly, the sphincter muscle, fascia and skin margin opposite the point of introduction (fig. 9). When this stitch is tied, the edges of the lower portion of the denuded area are closed thus approximating the rectal side of the perineal body segments. Two additional stitches are taken, one on each side. The other stitches are placed as was described in a first degree tear, (fig. 8).

In a third degree tear the outline is in the shape of a square with a triangle erected on the vaginal and rectal side (fig. 10: compare with fig. 6c). Instead of denuding the entire area, only a portion of the vaginal scar is denuded. The rectal portion is dissected from

above down to the pits on each side where the torn ends of the sphincter have been drawn. The ends of the sphincter are dissected up and sutured (fig. 10). The flap is then sutured to the ends of the sphincter muscle and perineal body segments and the skin margins on each side with a figure of 8 as was done in second degree

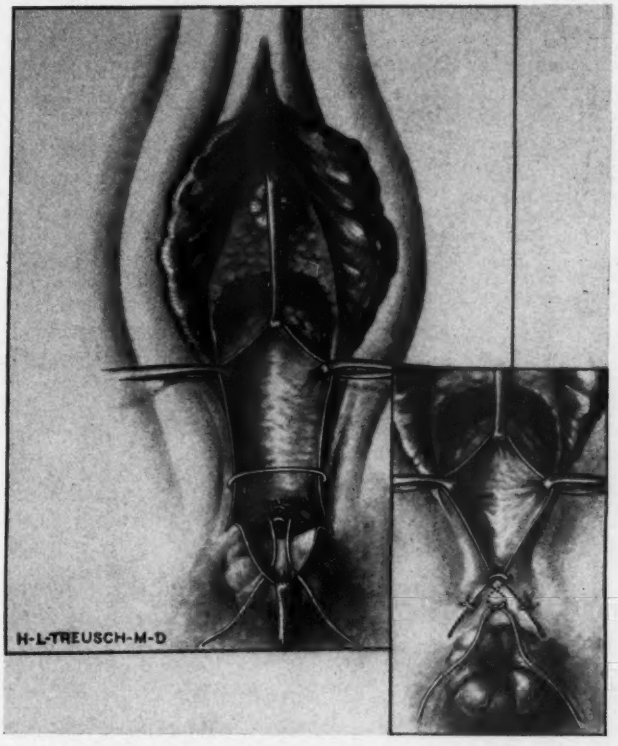

Fig. 11 (a-b). Indicates the placing of the figure of 8 suture for approximating the rectal side of the perineal body as was done in fig. 9. Inset shows the appearance of the wound after these sutures are tightened and tied. Other figure of 8 stitches are taken as was done in closing a first degree tear.

tears (fig. 11, a and b). Other sutures are placed as described before.

Comment

In the discussion of uterine supports, their arrangement, mechanics and correction of defects, no original information or technic is given. Only original diagrammatic drawings to illustrate the anatomy, physiology and mechanical arrangement of these supports is presented. In perineal supports, not only original diagrammatic drawings of the anatomic and mechanical arrangements are pre-

sented but also original technic in the surgical correction of defects is claimed, particularly that part which deals with the correction of perineal body tears and especially the placing of the figure of 8 suture in approximating the perineal body segments and closing the vaginal and skin wounds.

SUMMARY

1. The uterus is supported in a sling by mechanical supports.

2. Displacements of the uterus are due to altered mechanics of its supports.

3. The surgical correction of displacements is the restoration of the damaged supports to their original equilibrium.

4. The lower ends of the vaginal and rectal tubes are supported in a sling just as is the uterus.

5. The perineal body or central tendon which is placed between the ends of the vaginal and rectal tubes is the most important part of the perineal supports because it is subjected to greater hazards from trauma.

6. The perineal body and urogenital fascia are the supports usually damaged in perineal tears.

7. In the surgical correction of altered mechanics of the uterine or perineal supports only those structures which have been damaged should be repaired.

8. Original diagrammatic illustrations of the mechanical arrangement of both uterine and perineal supports and original technic in the correction of perineal supports are presented.

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ATLANTA

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TENTH ANNIVERSARY ASSEMBLY IN ATLANTA

The annual meeting of the Congress in March, 1939, marks the tenth birthday of the organization and we are anticipating the best meeting in its history.

In 1929, in Atlanta, a small group of surgeons, who felt that there was a definite need for a surgical organization in the Southeast, met and organized The Southeastern Surgical Congress. The first meeting was held in Augusta, in 1930. Since then annual scientific assemblies have been held in Birmingham, Atlanta, Nashville, Jacksonville, New Orleans, Charlotte and Louisville and now in 1939 it is fitting that we return to Atlanta, the city of its birth, for our tenth anniversary.

At each annual Assembly we have had some of the greatest surgeons and teachers of the nation on our program, and our attendance has increased with each Assembly, which demonstrates the need for this type of organization in the Southeast.

We began with seven states, we now have ten with a possibility of others. The purposes of this organization were well expressed in an editorial by our President-elect, Dr. R. L. Sanders, in THE SOUTHERN SURGEON, October, 1938:

The Southeastern Surgical Congress was conceived, developed and born into its present geographic setting by the hungry minds of a few Southern

surgeons who were eager for an opportunity to spread the gospel of better surgery in the South. The organization came into existence for the sole purpose of stimulating progress in medical science in the Southeastern part of the United States and neighboring territories. A few short months later the association was dedicated to stimulate progress in the science of surgery. Such an opportunity, such a privilege, such a responsibility, and such possible rewards! Even though still in its swaddling clothes, this association has made excellent progress, it has kept the faith and the rewards are to be seen dimly on the horizon of the future. Unborn generations of Southern surgeons may rise up to bless the hour of its birth and to give thanks unto the spirit of sturdy men who visualized the possibilities and responsibilities of such an organization.

Our annual Assembly is a regular postgraduate course including all of the surgical specialties, and this organization is credited with being the first all surgical postgraduate assembly in the United States. I wish here to pay tribute to our guest speakers who annually have come to us from the furthermost corners of the United States, and also to those speakers from our own territory, both of whom have made our programs the most outstanding surgical assemblies in the United States. While these meetings are for the specific purpose of stimulating surgical progress in the South, we want it definitely understood that the meeting is open to all physicians and we welcome them to our Assembly. We have a program which is well balanced and equal to the best. The scientific exhibits on the various phases of the art and science of surgery will themselves provide a postgraduate course. There will be a continuous moving picture exhibit showing the technic of various surgical operations and procedures, where one may go and actually see operations performed when he tires of hearing about them in the Auditorium.

Atlanta is located in the foothills of the Blue Ridge Mountains with an altitude of 1,100 feet above sea level with almost an ideal climate, it is the geographic center of the Southeast, as well as a railroad and air center. It has developed into a medical center since the War between the States, with the Medical School of Emory University located here, hundreds of doctors return annually to contact their Alma Mater and to attend medical conferences and postgraduate assemblies.

At this our homecoming meeting on our tenth anniversary, Atlanta welcomes you with open arms.

T. C. DAVISON, M. D., President.

Review *of* Neoplasms

formerly

The Review of Tumor Therapy

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CANCER SURGERY

There has been some question as to the causes of persistent swelling of the arm following radical amputation of the breast for carcinoma. Veal[1] made venographic and venous pressure studies on 46 cases and found that mechanical obstruction of the axillary and subclavian vein is the most frequent cause. This may arise from recurrence of malignancy, scar tissue formation, or angulation of the vein by fixation of the floor of the axilla. Simple lymphatic edema is infrequent but lymphatic stasis often occurs secondary to venous obstruction.

Carcinoma of the lung continues to receive much attention. Brock[2] reviews the English experience and emphasizes the value of exploratory thoracotomy in the presence of a mass of obscure nature with no reasonable alternative to a diagnosis of malignancy. He describes Edwards' technic for intrabronchial application of radon. Matz[3] notes the absolute increase of lung cancer among veterans and the high incidence of both preceding chronic diseases of the respiratory tract and occupational exposure to irritants. Strnad[4] in Prague found a high occupational exposure to smoke and dusts and family history of cancer in 40 per cent of cases. King[5] was able to make a positive diagnosis by bronchoscopy in 60 to 70 per cent of cases. Baum[6] describes a case of sarcoma diagnosed bronchoscopically where the clinical picture was that of atelectasis. Barrett[7] gives her technic for the examination of stained wet films of sputum for malignant cells and particles of malignant growth. In only 2 of 102 cases examined was a false positive report made. Huguenin[8] has written a timely paper on differential diagnosis. He describes various lesions of the pleura, mediastinum, and lungs which may be falsely diagnosed as carcinoma. Barrett and Elkington[9] give intercostal motor and sensory loss as diagnostic signs in endothelioma of the pleura.

The value of early diagnosis and early nephrectomy in kidney tumors is pointed out by Soloway[10] and by Ladd.[11] Wilm's tumor,

according to Ladd, has a good prospect for permanent cure if recurrence is not evident 1½ years after nephrectomy. Eleven of his 45 cases are living after 1½ to 19½ years. Renander[12] notes that the diagnosis of kidney tumor by pyelograms is not infallible. In 25 per cent of cases so diagnosed other conditions were found at operation. Congenital anomalies of the kidney are not often associated with malignancy. However, hypernephroma is reported arising in a double kidney by Ockerblad[13] and in a horseshoe kidney by Nicolich.[14]

Early carcinoma and hyperplasia of the cervix have been followed with biopsy studies by Scipiades and Stevenson.[15] The latent period is at least six months and may be six to eight years. They have observed that so-called precancerous hyperplasia may regress spontaneously in some cases. Todd[16] has studied rectal ulceration following irradiation treatment of carcinoma of the cervix. These late reactions may simulate carcinomatous involvement. The cause appears to be local overdosage in the vaginal vault which produces thrombosis of the hemorrhoidal vessels. Todd describes several technical details which may aid in avoiding this complication. Clivio[17] reports a case of adenocarcinoma arising in a uterus after irradiation for fibromyoma.

The place of gastroscopy in the diagnosis of both the presence and the operability of carcinoma of the stomach is reviewed by Schindler.[18] He states that an unfavorable diffusely infiltrating lesion can be recognized and a useless exploratory operation avoided. McNealy and Hedin[19] point out the frequency of perforation in cancer of the stomach. Perforation occurred 133 times, or 4 per cent of all cases. The operative mortality was 58 per cent and only 13 cases left the hospital improved. The operation of choice is simple closure primarily. They advise routine biopsy in all cases of gastric perforation, since many cases thought to be due to benign peptic ulcer may be malignant. Boettiger[20] found previous cholelithiasis in 80 per cent of cases of carcinoma of the gall bladder. Haines [21] reported a case of carcinoma of the head of the pancreas without jaundice. A spontaneous cholecystoduodenal fistula from old cholelithiasis had prevented signs of biliary obstruction, although the common duct was completely occluded.

Lockhart-Mummery[22] gives the technical details of his perineal excision for cancer of the rectum. Preliminary colostomy is performed about ten days before excision. Rankin[23] reports only 6.6 per cent mortality for the Miles operation in one stage for 75 recent cases. He performs the Lockhart-Mummery operation in about one fourth of cases.

Myerding[24] gives the results of treatment in 158 cases of osteogenic sarcoma. Tumors called grade 1 histologically showed 60 per cent five year survivals as compared to 10 to 15 per cent among those of higher grade malignancy. Local excision rather than amputation is done in grade 1 tumors.

Morton[25] discusses the value of a close association of the surgeon, radiologist and pathologist in the treatment of cancer; as best typified by the "tumor clinic" type of organization. The need for fundamental study and evaluation is pointed out; in order to establish the usefulness of various new methods. Sauerbruch[26] describes the use of combined surgical and radiation therapy in various types of cancer. Jackson and Jackson[27] discuss the indications for operation or irradiation in carcinoma of the larynx. In questionable cases the mobility of a lesion is a more reliable sign for operative removal than the degree of histologic differentiation.

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ADENOMAS OF THE COLON

Adenomas or adenomatous polyps are the commonest benign tumors of the intestinal tract.[1] They occur most frequently in the colon, particularly its left half, being eight times more common in the rectosigmoid and rectum than in any other section of the large bowel.[2] These tumors have been extensively studied by different observers using different approaches with the result that some confusion has followed various etiologic, histologic and clinical classifications. The term polyp itself is misleading usually being applied to any benign pedunculated or sessile tumor occurring within the lumen of the bowel. Thus lipomas, myomas, fibromas, papillomas, angiomas or argentaffine tumors, though histologically distinct, have been loosely classified in a general clinical group. The application of the term adenoma on morphologic grounds seems desirable. The classification of adenomas into adolescent (congenital or disseminated) and adult (acquired) types by Erdman and Morris[3] has been widely accepted. Wesson and Bargen[4] refer to the first group as true, the second as inflammatory adenomas. Lockhart-Mummery[5] considers the former a distinct familial disease due to a gene mutation inherited as a Mendelian dominant, for excessive proliferation of the epithelium of the colon at the age of puberty. Cases of infants, or babies born with the disease have not been noted, development being most common at puberty thus differing from most of the well known hereditary diseases. Meyer[6] and others have also regarded the epithelial reaction secondary to an underlying congenital malformation. The production of multiple adenomas following a long period of latency may possibly be explained by an over-responsive mucosal epithelium to repeated slight infections or to hormonal stimuli from endocrine changes first manifest at puberty.

The adult or acquired type of adenoma appears more definitely related to infections and it is not uncommon to find small peduncu-

lated tumors arising at the edge of healing amebic and tuberculous ulcers or following nonspecific chronic ulcerative colitis. Minute islands of mucous membrane become isolated by the inflammatory tissue of the healing ulcers with resulting outpouching and hyperplasia of the glands and ingrowth of new blood vessels. Feyrter[7,8] noted an increase in the incidence of adenomas with each advancing decade of life reaching an incidence of 77 per cent at the age of 75 and over. Among a total of 1,800 autopsies in all age groups the percentage incidence was 21.5 per cent. Whether chronic irritative or infective factors of long standing exerts a more profound influence in their production in old age groups cannot be answered. It is not unlikely on the other hand that many of the inflammatory reactions often noted in small adenomas are secondary changes rather than the initial reaction responsible for the proliferation of the mucosal glands. Trauma and active peristalsis frequently lead to erosion, hemorrhages and degenerative changes in adenomas preparing the soil for more extensive secondary inflammatory changes.

In both adolescent and acquired types of adenoma the basic pathologic structure is the same differing only in the degree of development. They may be sessile or pedunculated and vary in size from barely visible macroscopic mammillations to growths large enough to cause intussusception and obstruction. Microscopically groups of glands lined with cylindric epithelium of the type seen in Lieberkuhn's crypts, but with more basic staining cytoplasm, are supported by a connective tissue stroma derived from the submucosa. Mucus production is variable depending on local infection, nutrition and other factors.

The ultimate fates of these adenomas again differ somewhat in the congenital and acquired types though an exact evaluation of the percentage of malignant transformation in each group is offset by numerous difficulties. A history of congenital or familial distribution may be lacking; adenomatous polyps apparently increase in number in the higher age groups; marked variation in statistics occur depending on whether surgical or autopsy material is studied. Most observers,[11,12,15] however, place the incidence of malignant change in the congenital type adenomas from 40 to 60 per cent. Malignant changes, though common in the acquired type, are apparently considerably less frequent. Klemperer[10] studying the adult or acquired type tumors found 22.7 per cent of these adenomas occurring in colons resected for carcinoma showed malignant changes. The incidence of malignant changes in adenomas found in his autopsy material was 6 per cent.

Schmieden and Westhues[9] studied a large amount of surgically removed adenomas of the colon and classified them into three groups

attempting to establish histologic criteria for adenomas which were likely to become malignant. Group 1 included those covered with normal mucosa and were regarded as practically harmless while 2 and 3 were considered potentially malignant. Fitzgibbon and Rankin[13] made similar histologic investigations. However, such classifications are not without shortcomings and changes such as the number of mitoses, multilayering of cell nuclei, peripheral dark zones and variations in staining of cell cytoplasm may be found in perfectly benign adenomas, as well as forming an integral portion of adenomas with outspoken malignant changes. Klemperer denies the existence of any criterion which could be accepted as a reliable index for the malignant potentiality of any given adenoma and it would seem from the study of his and other material that such a conclusion is warranted. Nevertheless, the high percentage of malignant change occurring in both the congenital and acquired type and the absence of definite dependable cancerous histologic criteria require the surgeon to remove these tumors whenever encountered. Mayo and Butsch[14] discussing solitary adenomas of the colon advocate their immediate removal and report 5 cases showing definite adenocarcinoma grade 1 among 20 adenomatous polyps removed.

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THE PROBLEM OF ROENTGEN THERAPY

There are certain inherent factors of radio-resistance or sensitivity of tumors and the radio-tolerance of patients, both locally and generally, that we do not yet have the means of accurately or completely predetermining. Therefore, it is most essential to make use of, as completely as possible, all of the predeterminable physical and biologic factors at our disposal.

One of the most disconcerting things about radiation therapy is that in large series of malignancies, treated with totally different technics, the results appear to be equally good or perhaps we should say bad; for in such results the radio-sensitive tumors have undoubtedly responded, possibly in spite of the technics, and the resistant neoplasms have been unaffected. While many have mentioned it, as yet the necessity for individualizing treatments has not been sufficiently emphasized to take care of the majority of radio-resistant tumors. As an example of similar results with totally different methods of x-ray therapy Wintz[1] with his "one-time technic" (i. e., a single massive dose to one or more fields), has obtained about the same percentage of cures of cancer of the cervix uteri as have Coutard[2] and his followers with their protracted-fractionated treatments, which necessitate the irradiation of patients for literally hours each day, over periods of as long as five or six weeks, and eight to ten times the total accumulated dose which would be given with the Wintz technic.

It would seem unlikely that both of these technics are equally correct and yet there are elements of truth in both, otherwise neither would produce any favorable results. You can demonstrate this to your own satisfaction, if you have not already done so. With almost any skin epithelioma, of the basal or baso-squamous cell type, which is not more than a few millimeters deep or thick and not larger than a dime.

A radiation cure may be expected from a dose of 1,500 to 1,800 roentgens of unfiltered low voltage x-rays delivered at a rate of 5 to 100 r/mins. Also 300 r of the conventional deep therapy daily for twelve to thirteen days, at a rate of 10 to 75 r/mins., for a total dose of 3,600 to 3,900 roentgens will eradicate the lesion. With this latter technic there may be slightly less reaction but we cannot even be assured of this. I hope we can by analyzing various data, results and biologic facts begin to build up our own methods based on "individualized treatments"—which will embrace the good points of both extremes and will eliminate some of the shortcomings.

Read before The Georgia Radiological Society at Macon on Dec. 13, 1938.

The most objectionable feature from all angles of the Coutard technic I am glad at last can be quickly and summarily dealt with. The original contention that an extremely low intensity or small number of roentgens per minute (protraction) was necessary both to make the large total dosages possible and through this to assure the improved results that were being obtained has now been completely discredited. Such authorities as Ralston Paterson[3] of England, Weber[4] and Holfelder[5] in Germany, Borak[6] of Austria, Schinz[7] of Switzerland, Chamberlain and Young[8] in this country and countless others not only state that the low intensity is unnecessary but there seems to be a growing opinion that it may even be less effective than the usual intensities of modern therapy machines of 20 to 75 r/mins.

In the final analysis most modern therapy technics have as their common features high total dosages, an intensive course aimed at a cure not palliation and as homogeneous irradiation of the whole tumor as is possible, i. e., multiple ports which allow a greater tumor dose without over-irradiation of any particular volume or normal skin or tissue. The great discrepancies lie in the amount and extent of the fractionation and the spacing of the irradiation.

We may eliminate one other controversial feature before getting into the subject of spacing. It has been amply proven there is so little increase in the percentage depth dose at 400,000 or even 500,000 over the standard 200,000 volt technics (using 2 mm. of Cu or even less) with moderate sized or large fields, that to take advantage of higher voltages one must use a most accurately centered multiple port technic with field sizes not larger than 5 cm. by 5 cm. When a volume of tissue this small is irradiated the total number of rays in the primary beam are so reduced that the lessened secondary radiation (which, however, is probably as effective on the neoplasm as the primary rays) makes it possible to achieve a worth-while relative increase in the depth dose by increasing the voltage to 400,000 and therefore the effective penetrability of the decreased primary beam.

Since I gave as an example a skin cancer involving a very small quantity of tissue a good beginning for the discussion of the spacing of radiation is the relationship of the location and size of the growth and the amount of normal tissue that must be traversed to reach it. This brings up the all-important and usually neglected feature of the tissue tolerance of the patient. This tolerance is both acquired and inherent and we may dismiss acquired tissue tolerance by mentioning that local and general infections, age, sex, condition of the blood and many other factors which are understood and can be compensated for if not corrected will not only tend to affect the tolerance

of the normal tissues traversed by the rays but may also lessen the radio-sensitivity of the neoplasm.

The inherent tolerance of the patient, both locally and generally, is a much neglected subject and is, I believe, the main if not the only reason why similar results with the same type and location of internal cancer may be obtained with the "one-time" technic or protracted fractionation. Such varied technics may produce equally good results in certain cases because, as Holthusen[9] has recently pointed out, the tissue tolerance to x-rays in apparently normal patients may vary 300 per cent. In this event a radio-sensitive neoplasm in a subject with a high inherent tissue tolerance and without any localized acquired normal tissue susceptibility might have their cancer cured by a "blast" or massive dose of irradiation just as well as with a protracted-fractionated series.

However, the radio-sensitivity of many tumors is so little greater than the tolerance dose of the normal tissue that, when the tumor is deep lying it may be impossible to apply a cancericidal dose to the tumor, even with multiple ports, without fractionating the dose to each port.

In any technic one must apply the two following biologic principles to achieve an adequate tumor dose:

1. In a given period of time normal cells recover more completely from the effects of radiation than do even the mature type of malignant cells.

2. It is necessary to preserve the blood vessels to the tumor (at least for a while) since it has been proven by Coutard, clinically, and Mottram, experimentally, that cells with an insufficient oxygen supply are very radio-resistant. They are, of course, very inactive or quiescent but in the case of cancer cells they may lie dormant for indefinite periods of time yet when new blood vessels ultimately grow into the fibrous mass these dormant cancer cells may again resume their highly malignant character.

Evidence that with almost any type or degree of fractionation much larger dosages can be applied and a more decided effect produced in internal malignancy is so great that I will not attempt to enumerate references. There is considerable controversy as to who first demonstrated experimentally the increased effectiveness of fractionation on tumors with the concomitant decreased effect on the overlying normal tissue, i. e., the first biologic principle we have just mentioned, but Lacassagne and Regaud at the Curie Institute in Paris are usually given the credit. Now comes the second biologic principle of preservation of the vasculo-connective tissue or tumor bed. In fractional doses the radiation effect on the blood supply seems to parallel the skin reaction[10] while the effect on the cancer cells more closely approximates the total accumulated dose in roentgens. The greater the fractionation the more radiation can be de-

livered because of the recovery of normal tissues but if individual fractions are too small there is the danger that tumor cells will also recover. The objective aimed for is a sufficiently large fraction to prevent tumor cells from recovering before the next treatment and yet sufficiently small to keep from injuring seriously or permanently the normal overlying structures.

A secondary objective is not to apply radiation when the tumor is in a relatively radio-resistant phase thereby not subjecting the normal tissues to unnecessary radiation. Even Mottram[11] who is the pioneer in this work feels that this type of spacing of radiation is still in the experimental stage and he is at present using it only on tumors that can be seen or felt. I shall quote from an article:[12]

> It would seem that, at present, the only way to estimate radio-sensitivity during treatment, is to make frequent measurements of the tumours, and to assume that during the regression the tumour will be resistant and during growth sensitive, and to space the exposures accordingly. Some patients are being thus treated at this hospital. Even so, the problem is one requiring more experimental backing since, apart from spacing a second problem manifests itself; is it better to give a few large doses, say 500 r. or many small ones, say 100 r? It may be that during the application of a large dose, sensitivity falls so rapidly that the last 200 or 300 r are more or less wasted. In the present experiments it is evident that some of the 70-minute exposure is thus wasted since two exposures of 35 minutes, spaced 24 hours, are more effective.

In reported successful clinical applications his work[13] is being confined to external tumors but there is no reason why those interested in this important subject should not treat selected neoplasms in the mouth or cervix where frequent inspections, if not actual measurements, might allow one to judge when regression was no longer taking place; the neoplastic cells were again coming into their relatively radio-sensitive phase and it would be the time to apply further radiation. It seems to me that it might be possible to work out in advance a type of spacing from the number of embryonal or mitotic cells present in the slides being studied. For example, with a Type I Broder's Classification (0 to 25 per cent immature, abnormal, or mitotic cells) a different spacing would be necessary than for a Type IV Broder (75 to 100 per cent abnormal cells). Along with this tentatively planned spacing the actual reaction of the tumor and the patient would be used to individualize the treatments necessary. Various other radiologists have discussed the time of maximum tumor susceptibility but it is my impression, possibly an incorrect one, that it has all been rather inferential and deductive. However, there do not seem to be any theoretically insurmountable objections to working out this problem scientifically with the close cooperation of the radiologist, pathologist and clinician.

Since I have wandered rather far from the field of tissue tolerance and individualized treatment I would like to say a few more things about these before closing. Most important, as you already realize, they are so intimately associated that they may be considered as synonymous terms or, in other words, treatments have to be individualized to suit the patient's inherent tissue tolerance. For at least the last three years in our radiologic department, we have been extremely conscious of variations in tissue tolerance since we have overdosed some patients. In some extremely small females we have given too large quantities of x-rays when treating carcinoma of the cervix through a four field technic, even though the patient had no serious skin reactions, vesicovaginal or rectovaginal fistulae or other complications. The tissue seemed to be so devitalized that even though there was no demonstrable progression of the malignancy, in fact some attempt at healing took place, the patients went on and died, with symptoms and signs of an infection. The situation has been more frequently observed and reported after radium treatments.

We have perhaps been most successful in individualizing therapy where the neoplasm has been more superficial, as in carcinoma of the breast, larynx, etc. In these cases we frequently start with relatively high dosages say 300 r to each of two fields a day. If the patients will constitutionally stand this amount we will continue the same dosages but if not we reduce the roentgens per field but not the number of fields. After the total dose to a field has reached 1,200 to 1,500 roentgens we daily carefully inspect each treatment area and when the first "blush" appears, a suberythema effect, the treatments are discontinued since we have found that this invariably goes to a deep purplish erythema or patchy epidermolysis which we feel is normal tissue tolerance. As a rule in patients whose inherent tissue tolerance to radiation is low the radio-sensitivity of the neoplasm parallels this as the neoplasm is after all only a perversion of the patient's normal cells. If on the other hand we do not find that the neoplasm is regressing properly we find new areas to treat. In this respect the "multiple transfixation" technic advocated by Ffrangcon Roberts is extremely valuable. The following is quoted from his book:[14]

> In most cases where the tumour is small relatively to the area of skin around it, a large number of small fields should be used. In fact, the smaller the fields the better; they should certainly be smaller than a cross-section of the tumour, and should all be directed on the centre of the tumour. The advantage of small fields are as follows: First, each field being directed upon the centre of the tumour, the greatest dose falls upon the part where it is most required, that is, at the point of origin of the growth. Secondly, the body, as a whole, receives a smaller dose as compared with the large-field method, much of the

radiation by the latter method either being wasted or damaging the normal structures. Thirdly, there is a considerable economy in skin-fields. It is possible, for instance, to use nine fields of 3 x 3 cm. in the space occupied by one field of 12 x 12 cms., even allowing sufficient space to prevent overlap. If properly directed, the whole of each of the nine fields passes through the tumour, whereas a great part of the one field passes outside it.

By this method 400,000 volt therapy because of the small fields can be used to great advantage and will overcome the only real disadvantages of this method, which the author has mentioned himself.[14]

Two objections may be raised against small fields. The first is that the smaller the field the less is the penetration, owing to the fact that there is less scattered radiation. This is true, but at the same time the smaller the field the less is the skin affected by back-scatter. A small field can, therefore, tolerate a larger dose than a large field, this factor partly compensating for the loss in penetration. In any event the loss is a small matter in view of the large number of fields which are possible. A second possible objection is that insufficient use is made of the secondary radiation striking the tumour from the surrounding tissues. But what is lost in this way is more than compensated by the large number of fields.

Finally, I must again reiterate that this technic requires the greatest care and exactness and to end up on the thesis of individualization, *the technic is not applicable at all:*

1. If the tumor is large and widespread in comparison with the area of the skin surrounding it.

2. In postoperative, prophylactic irradiation, where the extent of possible disease is unknown.

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New Orleans

Dr. Ralph E. King
Winnsboro

MISSISSIPPI

SENIOR FELLOWS

Dr. J. F. Armstrong
Jackson

Dr. E. E. Benoist
Natchez

Dr. Maxwell D. Berman
Tower Bldg.
Jackson

Dr. J. A. K. Birchett, Jr.
Vicksburg Sanitarium
Vicksburg

Dr. T. H. Blake
414 Standard Life Bldg.
Jackson

Dr. John C. Culley
Oxford

Dr. John Darrington
Yazoo City

Dr. J. W. D. Dicks
Natchez

Dr. M. Q. Ewing
Amory

Dr. R. J. Field
Centreville

Dr. Ped L. Fite
Columbus

Dr. M. L. Flynt
Newton

Dr. A. E. Gordin
121 N. President St.
Jackson

Dr. F. H. Hagaman
Jackson

Dr. William F. Hand
519 Standard Life Bldg.
Jackson

Dr. R. D. Kirk, Jr.
Tupelo

Dr. I. C. Knox
Vicksburg

Dr. Lawrence W. Long
412 Standard Life Bldg.
Jackson

Dr. Hosea F. Magee
708 Lamar Life Bldg.
Jackson

Dr. Maury H. McRae
Corinth

Dr. Lonnie B. Moseley
Jackson Infirmary
Jackson

Dr. W. H. Parsons
Vicksburg

Dr. V. B. Philpot
Houston

Dr. Frederick Rehfeldt
Jackson Infirmary
Jackson

Dr. H. L. Rush
Meridian

MISSISSIPPI *(continued)*

Dr. L. V. Rush
Meridian

Dr. R. D. Sessions
Natchez

Dr. H. R. Shands
Jackson

Dr. R. M. Stephenson
Lexington

Dr. A. Street
Vicksburg

Dr. G. M. Street
Vicksburg

Dr. W. H. Sutherland
Booneville

Dr. J. S. Ullman
306 Franklin St.
Natchez

Dr. Frank E. Werkheiser
419 Standard Life Bldg.
Jackson

JUNIOR FELLOWS

Dr. Eugene A. Bush
Laurel

Dr. R. J. Field
Centreville

Dr. S. E. Field
Centreville

Dr. Toxey E. Hall
Miss. State Charity Hospital
Jackson

Dr. M. Murph Snelling
Gulfport

Dr. M. Brister Ware
512 Standard Life Bldg.
Jackson

NORTH CAROLINA

SENIOR FELLOWS

Dr. M. H. Biggs
Rutherfordton

Dr. A. G. Brenizer
Professional Bldg.
Charlotte

Dr. H. L. Brockmann
High Point

Dr. C. Z. Candler
Sylva

Dr. D. B. Cobb
Goldsboro

Dr. R. H. Crawford
Rutherfordton

Dr. J. W. Davis
Statesville

Dr. Crowell
Lincolnton

Dr. Robert T. Ferguson
Professional Bldg.
Charlotte

Dr. J. S. Gaul
Professional Bldg.
Charlotte

Dr. C. F. Glenn
Rutherfordton

Dr. T. V. Goode
Statesville

Dr. W. L. Grantham
807 Pub. Serv. Bldg.
Asheville

Dr. Parker C. Hardin
Monroe

Dr. V. K. Hart
106 W. Seventh St.
Charlotte

Dr. James D. Highsmith
Fayetteville

Dr. F. C. Hubbard
N. Wilkesboro

NORTH CAROLINA *(continued)*

Dr. R. O. Lyday
101 N. Elm St.
Greensboro

Dr. B. R. Lyon
Greensboro

Dr. W. F. Martin
608 Professional Bldg.
Charlotte

Dr. R .W. McKay
121 West 7th
Charlotte

Dr. R. B. McKnight
Professional Bldg.
Charlotte

Dr. Julian A. Moore
Flat Iron Bldg.
Asheville

Dr. C. S. Norburn
346 Montford Ave.
Asheville

Dr. H. H. Ogburn
Jefferson Bldg.
Greensboro

Dr. J. F. Robertson
Masonic Temple Bldg.
Wilmington

Dr. W. Paul Sanger
101 Medical Arts Bldg.
Charlotte

Dr. W. M. Scruggs
Professional Bldg.
Charlotte

Dr. W. H. Sprunt
403 N. Main St.
Winston Salem

Dr. C. V. Tyner
Leaksville

Dr. C. A. Woodard
Wilson

JUNIOR FELLOW

Dr. W. P. Holt, Jr.
Erwin

SOUTH CAROLINA

SENIOR FELLOWS

Dr. A. E. Baker
Baker Sanatorium
Charleston

Dr. Charles O. Bates
135 S. Main St.
Greenville

Dr. S. O. Black
392 E. Main St.
Spartanburg

Dr. H. S. Black
329 E. Main St.
Spartanburg

Dr. T. E. Bowers
89 Rutledge Ave.
Charleston

Dr. W. A. Boyd
1500 Washington St.
Columbia

Dr. A. J. Buist, Jr.
279 Meeting St.
Charleston

Dr. Johnston Buist
279 Meeting St.
Charleston

Dr. George H. Bunch
1404 Laurel St.
Columbia

Dr. A. F. Burnside
1318 Lady St.
Columbia

Dr. Francis G. Cain
4 Vanderhorst St.
Charleston

Dr. J. W. Corbett
Camden

SOUTH CAROLINA *(continued)*

Dr. T. M. Davis
204 N. Main St.
Greenville

Dr. R. G. Doughty
1417 Hampton St.
Columbia

Dr. C. B. Earle
135 S. Main St.
Greenville

Dr. C. B. Epps
Sumter

Dr. Charles H. Fair
Professional Bldg.
Greenville

Dr. J. M. Fleming
Spartanburg

Dr. L. C. Hays
Clinton

Dr. P. G. Jenkins
66 Wentworth St.
Charleston

Dr. Douglas Jennings
99 Market St.
Bennettsville

Dr. C. J. Lemmon
132 N. Washington
Sumter

Dr. F. H. McLeod
Florence

Dr. J. C. McLeod
Florence

Dr. L. M. McMillan
Mullins

Dr. D. L. Maguire
187 Calhoun St.
Charleston

Dr. C. A. Mobley
Orangeburg

Dr. A. T. Moore
Gervais & Pickens
Columbia

Dr. J. J. Ravenel
96 Ashley Ave.
Charleston

Dr. Cecil Rigby
Andrews Bldg.
Spartanburg

Dr. J. S. Rhame
81 Westworth St.
Charleston

Dr. James A. Sasser
Conway

Dr. C. J. Scurry
Greenwood

Dr. S. B. Sherard
Gaffney

Dr. J. R. Sparkman
221 E. Main St.
Spartanburg

Dr. J. E. Smith
60 Meeting St.
Charleston

Dr. L. P. Thackston
Orangeburg

Dr. W. P. Turner
310 Maxwell Ave.
Greenwood

Dr. G. T. Tyler
605 E. North St.
Greenville

Dr. Milton Weinberg
132 N. Washington St.
Sumter

Dr. J. Warren White
204 S. Main St.
Greenville

Dr. J. R. Young
126 E. Earle St.
Anderson

JUNIOR FELLOWS

Dr. Herbert Blake
130 E. Benson St.
Anderson

Dr. John H. Cathcart
E. Frederick St.
Gaffney

SOUTH CAROLINA *(continued)*

Dr. Henry Y. Harper
Professional Bldg.
Anderson

Dr. Frank A. Hoshall
95 Rutledge Ave.
Charleston

Dr. George S. Rhame
Camden

TENNESSEE

SENIOR FELLOWS

Dr. William M. Adams
902 Madison Ave.
Memphis

Dr. Herbert Acuff
603 W. Main Ave.
Knoxville

Dr. Shields Abernathy
1001 Madison Ave.
Memphis

Dr. Troy P. Bagwell
807 Medical Arts Bldg.
Knoxville

Dr. J. W. Bodley
915 Madison Ave.
Memphis

Dr. Perry Bromberg
Bennie Dillon Bldg.
Nashville

Dr. Willis C. Campbell
869 Madison Ave.
Memphis

Dr. L. E. Coolidge
Greenville

Dr. Kyle C. Copenhaver
902 Medical Arts Bldg.
Knoxville

Dr. Jewell M. Dorris
899 Madison Ave.
Memphis

Dr. I. G. Duncan
Bank of Commerce Bldg.
Memphis

Dr. Carey O. Foree
Foree Hospital
Athens

Dr. William E. Foree
Foree Hospital
Athens

Dr. Joseph H. Francis
Exchange Bldg.
Memphis

Dr. George Gartly
Varnsworth Bldg.
Memphis

Dr. W. D. Haggard
Doctors Bldg.
Nashville

Dr. R. N. Herbert
Medical Arts Bldg.
Nashville

Dr. Victor Hill
202 Doctors Bldg.
Knoxville

Dr. A. G. Kern
Box 405
Knoxville

Dr. Marion S. Lombard
U. S. Marine Hospital
Buffalo, N. Y.

Dr. Carroll H. Long
Johnson City

Dr. J. W. McClaran
Jackson

Dr. J. L. McGehee
508 Physicians & Surg. Bldg.
Memphis

Dr. T. C. McNeer
Kingsport

Dr. A. D. Mason, Jr.
Exchange Bldg.
Memphis

TENNESSEE *(continued)*

Dr. E. D. Mitchell, Jr.
Bank of Com. Bldg.
Memphis

Dr. T. D. Moore
899 Madison Ave.
Memphis

Dr. E. T. Newell
707 Walnut St.
Chattanooga

Dr. Jarrell Penn
Medical Arts Bldg.
Knoxville

Dr. J. C. Pennington
700 Church St.
Nashville

Dr. H. Dewey Peters
Med. Arts Bldg.
Knoxville

Dr. W. T. Pride
1460 Madison Ave.
Memphis

Dr. E. L. Rippy
Bennie Dillon Bldg.
Nashville

Dr. R. L. Sanders
324 Physicians & Surg. Bldg.
Memphis

Dr. R. E. Semmes
899 Madison Ave.
Memphis

Dr. D. C. Seward
Bennie Dillon Bldg.
Nashville

Dr. J. S. Speed
869 Madison Ave.
Memphis

Dr. Albert Sullivan
2318 West End Ave.
Nashville

Dr. Morton J. Tendler
899 Madison Ave.
Memphis

Dr. Raymond Wallace
1st Nat'l. Bank Bldg.
Chattanooga

Dr. Richard G. Waterhouse
401 Med. Arts Bldg.
Knoxville

Dr. P. H. Wood
Col. Mutual Tower
Memphis

Dr. M. C. Wiggins
Paris

JUNIOR FELLOWS

Dr. Ernest G. Kelly
899 Madison Ave.
Memphis

Dr. Hillard W. Otey
899 Madison Ave.
Memphis

Dr. Percy B. Russell, Jr.
915 Madison Ave.
Memphis

Dr. W. T. Satterfield
919 McLemore
Memphis

Dr. Robert C. Taylor
899 Madison Ave.
Memphis

VIRGINIA

SENIOR FELLOWS

Dr. C. J. Andrews
Medical Arts Bldg.
Norfolk

Dr. Horace G. Ashburn
19 Springfield Ave.
S. Norfolk

VIRGINIA *(continued)*

Dr. Elisha Barksdale
Allied Arts Bldg.
Lynchburg

Dr. Philip W. Boyd
212 W. Boscawen St.
Winchester

Dr. Claude C. Coleman
503 Professional Bldg.
Richmond

Dr. J. D. Collins
Professional Bldg.
Portsmouth

Dr. C. J. Devine
229 W. Bute St.
Norfolk

Dr. Benjamin A. Doggett
708 Medical Arts Bldg.
Norfolk

Dr. Beverley F. Eckles
Galax Hospital
Galax

Dr. Lomax Gwathmey
220 W. Freemason St.
Norfolk

Dr. Frank S. Johns
Johnston-Willis Hospital
Richmond

Dr. J. Coleman Motley
Abingdon

Dr. Julian L. Rawls
Medical Arts Bldg.
Norfolk

Dr. Alexander M. Showalter
Christiansburg

Dr. C. Carroll Smith
142 W. York St.
Norfolk

Dr. Hugh H. Trout
Jefferson Hospital
Roanoke

Dr. W. R. Whitman
Lewis-Gale Hospital
Roanoke